A DISSERTATION

ON

"STUDY OF COMPARISON OF HAEMATOLOGICAL MANIFESTATION IN ALCOHOLICS WITH NONALCOHOLICS''

Submitted to

THE TAMILNADU DR. M. G. R UNIVERSITY
CHENNAI

In partial fulfilment of the regulations
for the award of

M.D DEGREE IN GENERAL MEDICINE
BRANCH I

GOVERNMENT MOHAN KUMARAMANGALAM

MEDICAL COLLEGE, SALEM

APRIL 2016

Government Mohan Kumaramangalam Medical College Hospital

SALEM

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled **"STUDY OF COMPARISON OF HAEMATOLOGICAL MANIFESTATION IN ALCOHOLICS WITH NONALCOHOLICS"** is a bonafide and genuine research work carried out by me under the guidance of

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Acknowledgement

I would like to acknowledge **Mr Nandhakumar**, for helping me to analyze and compile the statistical data for my study.

I extend my heartfelt thanks to all my colleagues and friends for their help rendered during my study.

I specially thank all my patients without whose cooperation; this dissertation would never have seen the light of the day.

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Ethical Committee Meeting held on 30.07.2014 at 12 noon in the Dean's Chamber, Government Mohan Kumaramangalam Medical College Hospital , Salem 01, The following Members were attended the meeting .

MEMBERS.

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9.	Dr.S.Myilsamy √ Final year MD(GM)., Post Graduate Student GMKMC Salem 30	Study of Prospective study of Comparative Study of Haematological Manifestation in Alcoholics and Nonalcoholics 100 Cases in GMKMCH	Dr.S.Ramasamy MD., Professor of General Medicine	He is advised to Measure the quantity of Alcohol in ML. and also he is advised to measure Serum Folic Acid levels in the Patients Approved

The Ethical Committee examined the studies in detail and is pleased to accord Ethical Committee approval for the above Post Graduate of this College to carry out the studies with the following conditions.

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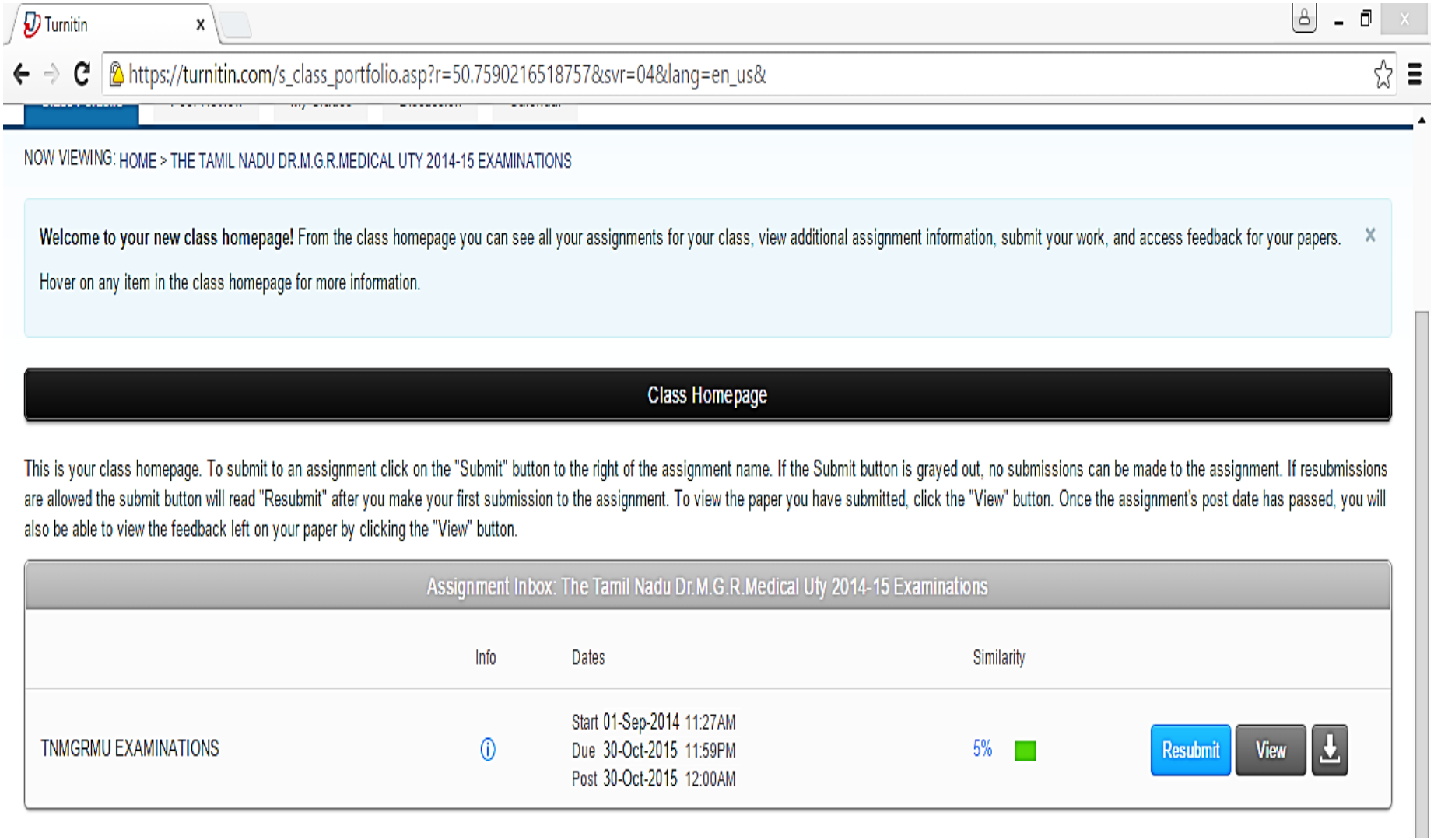
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LIST OF ABBREVATIONS

MCH	**Mean Corpuscular Haemoglobin**
MCV	**Mean Corpuscular Volume**
RBC	**Red Blood Cells**
ADH	**Alcohol Dehydrogenase**
TNF	**Tumour Necrosis Factor**
MEO	**Microsomal Ethanol Oxidising**
ATP	**Adenosina triphosphatase**
IL	**Inter Leukin**
WBC	**White Blood Cells**
TC	**Total Count**
MCHC	**Mean Corpuscular Haemoglobin Concentration**
Hb	**Haemoglobin**
PCV	**Packed Cell Volume**
SAM	**S-Adenosyl Methionine**
SAH	**S-Adenosyl Homocysteine**
CYP	**Cytochrome – P**
TGF	**Transforming Growth Factor**
ROS	**Reactive Oxygen Species**
BFU	**Burst Forming Unit**
CFU	**Colony Forming Unit**
ALA	**Amino Levulanic Acid**

AST	**Aspartate transaminase**
ALT	**Alanine Transaminase**
PT	**Prothrombin Time**
GGT	**Gamma Glutamyl Transferees**
AUD	**Alcohol Use Disorder**

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ABSTRACT

INTRODUCTION:

Alcoholism is one of the most serious global health problem. Incidence of alcoholism is high especially in lower socio economic groups of people and its affects all the systems like hepatobiliary system, cardio vascular system, central nervous system, haemopoietic system. Many a times haematological changes are left undetected and untreated which could progress to cardiac failure. Alcohol can lead to all types of anaemia with bone marrow suppression.

OBJECTIVES:

1. To study the haematological changes in alcoholics and non-alcoholics.
2. To study the haematological changes with respect to the quantity and duration of alcohol consumption are divided into moderate and severe drinkers.

MATERIALS AND METHODS:

25 adult patients who are moderate alcoholics, 25 adult patients who are severe alcoholics and 50 adult patients of non-alcoholics were included in the study in government Mohan Kumaramangalam medical college hospital Salem.

RESULT:

In our study 76% of severe alcoholics are anaemic and 72% of moderate alcoholics are anaemic. MCV of more than 99fl is present in 28% of severe drinkers and 8% of moderate drinkers. Mean Hb in severe drinkers is 9.37± 2.30.

Total count mean value in moderate alcoholics is 7872 ± 3537.72 and in severe alcoholics is 11072 ± 4286.17. Mean platelet count is 1.63 ± 0.66 and 1.47 ± 0.67 in moderate and severe drinkers respectively.

Mean folic acid value in moderate alcoholics is 8.71 ± 1.00 and in severe alcoholics is 8.08 ± 1.01. Mean value of vitamin B12 is 713.80 ± 374.14, 1375.60 ± 510.03 in moderate and severe drinkers respectively. Microcyte hypochromic anaemia due to iron deficiency is common among alcoholics followed by macrocytic anaemia due to folate deficiency, not by B12 deficiency.

Abnormal RBC morphology like target cells, acanthocytes, stomatocytes, elliptocytes and ovalocytes are seen in alcoholics.

CONCLUSION:

Alcoholism is present in both males and females and predominantly seen in males especially in lower socio economic groups in 3^{rd} to 5^{th} decade. Anaemia is common among chronic drinkers and its severity is related to amount and duration of alcohol consumption. Infections are common among severe alcoholics. Microcytic hypochromic anaemia is common due to iron deficiency followed by macrocytic anaemia due to folate deficiency. Thrombocytopenic is also common among alcoholics. Abnormal RBC morphology like stomatocytes, target cells, acanthocytes are seen.

Detection of haematological changes in alcohol abuse early and treating them will prevent alcohol related complications in future and decrease the morbidity and mortality in alcoholics.

KEY WORDS:

Anaemia; thrombocytopenia; erythrocytes; bone marrow; alcoholism; moderate and severe drinkers; haematological profile.

Introduction

INTRODUCTION

Alcoholism is one of the most serious global public health problem. Regarding disease Burden Alcohol is the world's third largest risk factor.

Alcoholism (Alcohol use disorder) is defined as repeated alcohol related difficulties in atleast 2 of 11 life areas that cluster together in the same 12 months period[1]. Lifetime risk for an AUD in most of the western countries is about 10-15% for men and 5-8% for women. Approximately 60% of the risk for AUD is attributed to genes.

Alcoholism results in 3.3million deaths per year globally. AUD decreases the life span by 10 years.

The factors increasing risk of liver disease in alcoholics are quantity and duration of intake, Sex (Females susceptibility twice as males), co-infection with Hepatitis C, Genetic factors, Malnutrition, Obesity, Smoking and Iron over load.

Two Billion people consumes alcohol Worldwide, as estimated by WHO. Among these 76.3 million people have AUD. Incidence of alcoholism is high especially in lower socio-economic status groups and they prefer low cost beverages with less quality and more deleterious effects on the organ system.

Hence alcohol consumption is known for morbidity and mortality, being a serious health hazard of the world. Multiple organs can be involved like Hepatobiliary system, cardiovascular system, Central Nervous system, Haemopoietic system. Many a times Haematological changes are left undetected and untreated which could progress to Cardiac failure.

Our study is to describe the Haemotological changes in alcoholics and to compare with non-alcoholics. Early detection and treatment of haematological changes can prevent complications and reduce the mortality, these are the basis and the need for the study.

Also early detection of alcohol abuse with the help pf haematological profile can prevent alcohol related disease in future like Cirrhosis Liver, Cardiac failure, Renal disease, Cerebellar atrophy, Peripheral neuropathy, Pancreatitis, tuberculosis, etc. by giving psychiatric counselling and treatment for alcohol dependence, there by morbidity and mortality due to alcoholism will be reduced.

Aims and Objectives

AIMS AND OBJECTIVES

- To study the haematological manifestation in alcoholics.
- To study the haematological manifestation in moderate and severe alcoholics based on quantity and duration of alcohol intake.
- To compare the haematological manifestations in alcoholics and non-alcoholics.

Review of Literature

REVIEW OF LITERATURE

Different types of Alcoholic beverages have been consumed globally for thousands of years. Excess intake of alcohol accompany lot of problems like physical, mental illness and social distress undoubtedly. So alcohol has ambivalent position always in the community.

However moderate intake of alcohol, especially red wine is beneficial to health, as it reduces LDL cholesterol and inhibit platelet aggregation, thus prevents coronary events.

Undeniably, alcohol is most widely used mood-altering agents in our community and cultural activities. From a psychological point of view, alcohol is dangerous abusing drug.

Recent changing trends like greater social acceptability in alcohol consumption greatly increases the number of consumers especially in youth population to overcome social Inhibitions in shy person, to produce sense of wellbeing and pleasure.

However, there is debate whether alcohol is consumed because of these psychological or medical effects, or associated with positive social interactions youthfulness and sexuality.

Religious campaigns and constitutional amendments has been failed to ban alcohol use. American journal **defines alcoholism as "a primary chronic disease characterized by impaired control over drinking, pre occupation with the drug alcohol, use of alcohol despite adverse consequences and distortions in thinking"[2].**

Americans has right to drink as the bill of rights.

Chronic alcohol abuse results in physical illness, emotional changes and others in their lives and relationships. If alcoholism is not treated, it causes premature deaths through damage to liver, brain, kidney, heart and other organs also. Alcohol dependence is one of the leading contributor to road traffic accidents, suicidal attempts, violence and other events. Untreated alcohol dependence people will lose their jobs, families and other relationships.

The extent of changes tends to increase and often worsens the life situations.

Alcohol problems can often be prevented by early identification and brief intervention. The weak links is identifying problems early are the skill and competencies necessary for such an assessment and the experience to confidently move to more specific questions and suggestion for change.

Completing this program will help the physician update his or her skill set and master the care issues in moderate alcohol consumption and

in alcohol abuse, problems and dependence detection as well as intervention.

HISTORY OF ALCOHOL

The exact date of alcohol production for first time remains elusive, the intentionally fermented beverages existed as early as the Neolithic period[3]. Around 4000 B.C. in Egyptian pictographs, wine has appeared in China, different types of alcoholic beverages have been used since prehistoric times[3].

Beer was one of the major beverages considered by the Babylonians. The Sumerians have the art of beer making pictorially. Around 2000 B.C[3]. the art of making wine reached the Hellenic Peninsula.

Alcoholic beverages were an intricate part of most civilization ranging from India and China and Western Asia to Europe. Around 800 B.C[3]. in India and China, distilled spirits was originated. In the eleventh century. In Europe distillation process emerged.

The God Bacchus was worshipped by the Greeks as the God of wine and the same God worshipped by Romans under a different name Dionysus. Beer was introduced by Romans to the Europeans around 55 B.C[3].

After rise of Christianity, wine became a part of rituals and there was a spread of Christianity in Europe simultaneously. Wine reached the Americans from Europeans and by this time Americans were developing their own versions[3].

Wine was considered as the best beverage during the middle ages and alcohol consumption began to spread globally[3].

ALCOHOL TYPES AND EFFECTS

Alcohol is colourless and flammable liquid. It is obtained by sugar and starch fermentation. It is used either in its pure form or denatured form. It is used as an explosives, Intoxicating beverages, cleaning solutions or as a solvent in drugs.

Alcohol is classified based on the number of carbon atoms in it. Methyl alcohol and Ethyl alcohol are most commonly used primarily. Methanol is also called as wood alcohol, Since it is obtained from wood distillation.

Ethyl alcohol is used in alcoholic beverages preparation and as industrial solvent. Methylated spirits or surgical spirits are form of ethyl alcohol. Isopropanol is a simple secondary alcohol and the simplest tertiary alcohol is Butanol.

The primary alcohols like Methyl alcohol and Ethyl alcohol are used in industries as fuels, solvents and reagents. These will burn more

effectively than diesel and gasoline. Ethyl alcohol is used as a solvent in perfumes also.

Alcohol will be obtained by fermentation of fruits or grains with yeast. Ethyl alcohol is produced through process of fermentation in the fuel production process. Humans consuming Ethyl alcohol since prehistoric times in the form of alcoholic beverages.

Consumption of moderate amount of alcohol is beneficial to heart, since it increased HDL cholesterol and inhibit platelet aggregation and thrombus formation, decreases the risk of Coronary Artery Heart Disease. Also moderate intake will prevent Alzhiemer's disease.

Consumption in large doses as binge drinking causes pancreatitis, alteration in Hematological profile, CNS depression and acute respiratory failure. Chronic consumption of alcohol in large amount leads to liver disease, anemia, peripheral neuropathy, cerebellar degeneration, heart failure and renal disease.

Twenty percent of consumed alcohol is absorbed in stomach and remaining 80 percent by small bowel. Chronic alcohol consumption also leads to recurrent falls, Road traffic accidents, Osteoporosis and Fractures, Nutritional deficiencies like Thiamine, Iron, Folate, Zinc frequent infections like Tuberculosis, CNS Infections and malignancies.

A person who consuming alcohol initially experience a stage of Euphoria followed by stage of Excitement, sense of stupor and finally leads to coma or death, when consumed in excess quantity.

Alcohol causes lot of problems in community like violence, poverty, crime, alcoholism, Family disintegration and disease burden. The emotional disturbances charged a person towards the attitude of drinking, drunkenness and alcoholism. These attitude are due to many factors, Such as family situation, biological differences, differing religious beliefs, socio cultural experience, prohibition, political, economic and personal feelings.

GENDER DIFFERENCE

The gender difference in alcohol consumption shows extreme patterns. Prevalence in women is less than 5 percent but much higher in northeastern states of India[6].

Recent trends in alcohol consumption changed in India that increased drinking among women due to ready availability, decreased social stigma regarding drinking. Initiation age also decreased with shift from urban to rural areas and pattern of binge drinking[6].

High alcohol abuse pattern seen in tribal and rural people and lower socioeconomic people in urban areas[6].

One fourth of the family income is spent on alcohol, and there is increased suicidal tendencies, domestic violence and affects the work concentration. One study conducted by the National Institute of Mental Health and Neuro Sciences, Bangalore. Shows that 20 percent of domestic violence reported by women and 94.5 percent of domestic violence is due to alcohol consumption[5,6].

Though alcohol consumption is increased among women, alcohol abuse is less among them. They are more prone to liver injury with doses less than that of males. Relapse rate after treatment of alcoholic hepatitis is high and fastly progress to cirrhosis from hepatitis stage, even with abstinence[6].

EPIDEMIOLOGY

Alcoholism represents one of the most serious worldwide socio-economic health problem.

INDIAN SCENARIO:

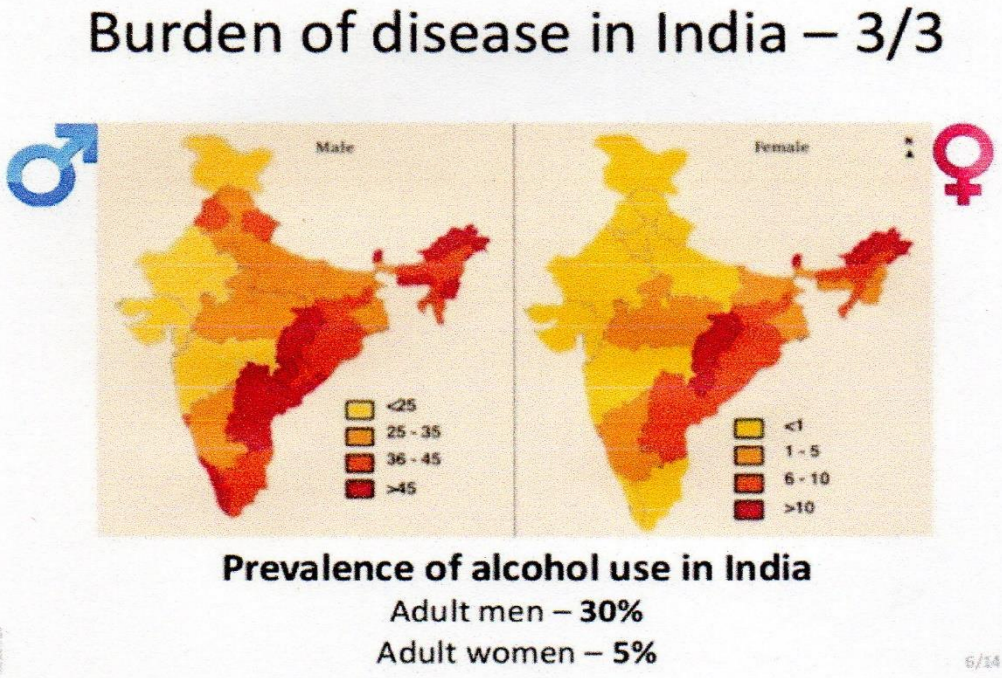
According to WHO, the alcohol consumption rate increased during the year 2008 – 2012, when compared with previous years. In India 30% of population consuming alcohol. In alcoholics 93% of people consuming hard liquor such as Whisky, Vodka and Spirit. 7% of them drinking Beer and only 1% alcoholics drinks Wine. Low socio-economic groups of people consuming low cost and low quality drinks.

Above 15 years of age, 32% of men and 11% of women consuming alcohol. Per capita alcohol consumption in 2003 – 2005 was 1.6 litre and it was increased to 2.2 litre during the year 2010 – 2012.

An average Indian male drinkers over 15 years of age drinks 33 litres of alcohol per year and women drinks 11 litres per year, according to WHO. In India, Kerala stands first in alcohol consumption an average individual of more 15 years of age drinks 8 litres of alcohol per annum. Next to Kerala, Maharashtra and Punjab leads.

11% of Indian population are involved in Binge drinking.

Figure: 1



GLOBAL SCENARIO

38.3% of population consuming alcohol globally. Every person of more than 15 years of age consuming alcohol at a rate of 6.2 litres per year. An average drinker, globally consumes 17 litre / annum. 16% of population included in binge drinking. Globally Europe is in first place in alcohol consumption.

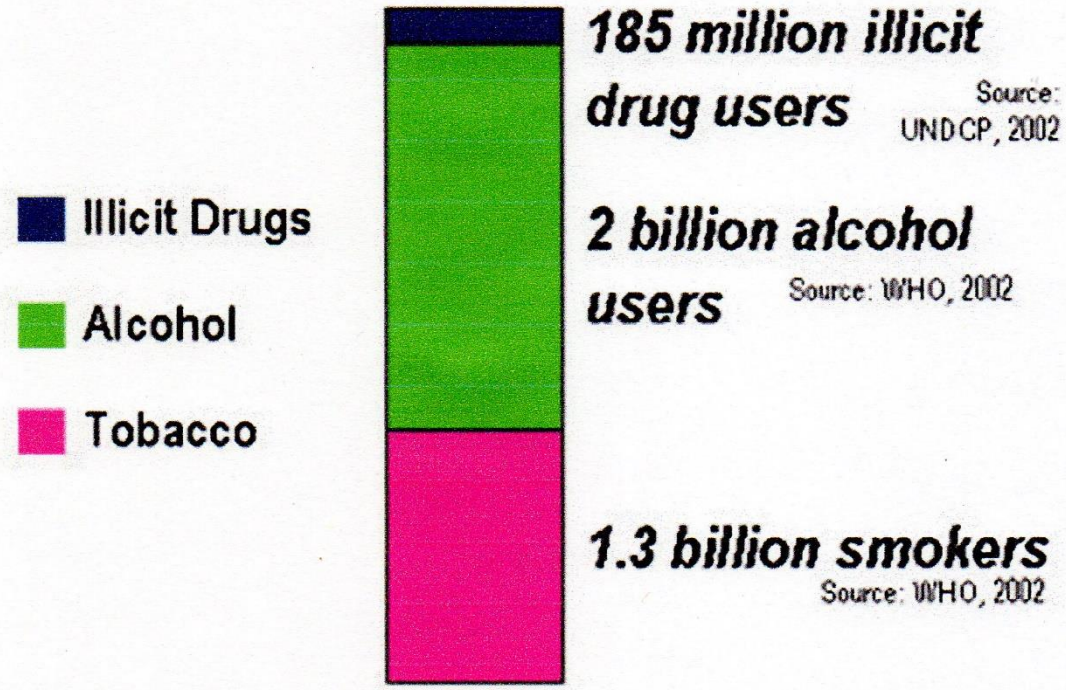
According to WHO, 2012 data 3.3 million death every year is due to alcohol abuse (5.9% of all death). In the age group of 20 – 39 years 25% of men died and 4% of women died due to alcohol consumption. Per capita alcohol consumption in Americans are 8.5 – 9.9 L annually and in Canadians 12.5 litre/ annum.

Alcohol consumption also contributes to about 10% of the disease burden due to tuberculosis, epilepsy, haemorrhagic stroke and hypertensice heart disease.

In India "number of years of life lost" scale rated 4.

Global Incidence of Alcoholism

Figure: 2



RECENT TRENDS IN ALCOHOL CONSUMPTION PATTERNS IN INDIA

1. Emergence of wine and beer drinking.
2. Increased drinking among women.
3. Decreased age of initiation.
4. Shift from urban to rural areas.
5. More binge drinking.
6. Greater social acceptability.

DRINKING PATTERNS

Daily intake of 160g/day of alcohol for 8 years will lead to cirrhosis .Intake of less than 160 g/day will leads to alcoholic Hepatitis in 40% of patients. The extent of liver damage depends on content of alcohol, not on type of beverages.

Intermittent consumption is safer than daily consumption because of chance of recovery for the liver. Daily intake of 160g of ethanol for less than 5 years will not cause hepatitis or cirrhosis[6].

NUTITION

Alcoholic supplies calories (a drink will give 70-100 kcal) devoid of nutrition. It will cause protein depletion and deficiency of vitamins

such as Thiamine, pyridoxine, Folate, Vitamin A and Nicotinic acid and minerals like zinc, iron etc.,

DEFINITION

One drink is defined as 12 ounces of beer, 4 ounces of wine, 1.5 ounces of hard liquor (Whisky, Gin, Rum, Vodka, Tequila)

One standard drink is equal to 14 gms of pure alcohol approximately. 1 fluid ounce is equal to 30 millilitre.

Moderate Alcohol Consumption:

Moderate drinking is defined as equal to or less than two drinks a day for men and one drink or less a day for women.

Heavy Alcohol Consumption:

- ✓ Hazardous or Heavy drinking is defined as more than 14 drinks per week or four to five drinks on any day at one sitting for men.
- ✓ More than seven drinks per week or 3 drinks on any day at one sitting for women.
- ✓ Frequently intotication.

Binge Drinking:

- Binge drinking is defined as consumption of five or more drinks for men and more than four for women within two hours.
- Blood alcohol concentration is 0.08g/dl.

Table-1

ALCOHOL CONTENT IN VARIOUS BEVERAGES:[8]

Beverage	Alcohol content	Serving Size		Amount of Alcohol	Daily Intake Needed to Exceed Threshold for Alcoholic Liver Disease	
		Oz	ml		Men	Women
Beer	5%	12	360	13.85gm	3-6 cans 1080ml – 2160ml	1.5-3 cans 540ml – 1080ml
Wine	12%	4	120	10.7gm	4-8 glasses 480ml – 960ml	2-4 glasses 240ml – 480ml
Fortified Wine	20%	4	120	17.8gm	2-4 glasses 240ml – 480ml	1-2 glasses 120ml – 240ml
Hard Liquor (Whisky, Gin, Rum, Vodka, Tequila, etc.)	40%	1.5	50	13.4gm	3-6 drinks 150ml – 300ml	1.5-3 drinks 75ml – 150ml

Table:2
Amount of Alcohol consumption in moderate and severe drinkers:

Beverage	Moderate drinking		Severe Drinking			
	Male	Female	Alcohol ml/day		In one sitting in a week	
			Male	Female	Male	Female
Beer	≤720 ml	≤360 ml	>720 ml	>360 ml	1.44 L	1.08 L
Wine	≤320 ml	≤160 ml	>320 ml	>160 ml	640 ml	480 ml
Fortified Wine	≤200 ml	≤100 ml	>200 ml	>100 ml	400 ml	300 ml
Hard liquor (Whisky, Gin, Rum, Vodka, Tequila, etc.	≤100 ml	≤50 ml	>100 ml	>50 ml	200 ml	150 ml

DIAGNOSIS OF ALCOHOL ABUSE

1. History
2. AUDIT and CAGE questionnaire.
3. Laboratory Diagnosis by
   a) Gamma glutamyl transferase (>35 Unit)
   b) Carbohydrate deficient transferrin (CDT >20 Unit/L)

4. MCV of more than (or) equal to 91 fl.
5. Serum uric acid of more than 7 mg/dl.

Table: 3

THE ALCOHOL USE DISORDERS IDENTIFICATION TEST (AUDIT)

ITEM	5 POINT SCALE
1. How often do you have a drink containing alcohol?	Least to most never (o) to 4+ per week (4)
2. How many drinks containing alcohol do you have on a typical day?	1 (or) 2 (o) to 10+ (4)
3. How often do you have six or more drinks on one occasion?	Never (o) to daily or almost daily (4)
4. How often during the last year you found that you were not able to stop drinking once you had started?	Never (o) to daily or almost daily (4)
5. How often during the last year have you failed to do what was normally expected from you because of drinking?	Never (o) to daily or almost daily (4)
6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session.	Never (o) to daily or almost daily (4)
7. How often during the last year have you had a feeling of guilt or remorse after drinking?	Never (o) to daily or almost daily (4)
8. How often during the last year have you been unable to remember what happened the night before because you had been drinking?	Never (o) to daily or almost daily (4)
9. Have you or someone else been injured as a result of year drinking?	No (o) to yes, during the last year (4)
10. Has a relative, friend, doctor or other health worker been concerned about your drinking or suggested that you should cut down?	No (o) to yes, during the last year (4)

Table: 4

CAGE – Questionnaire

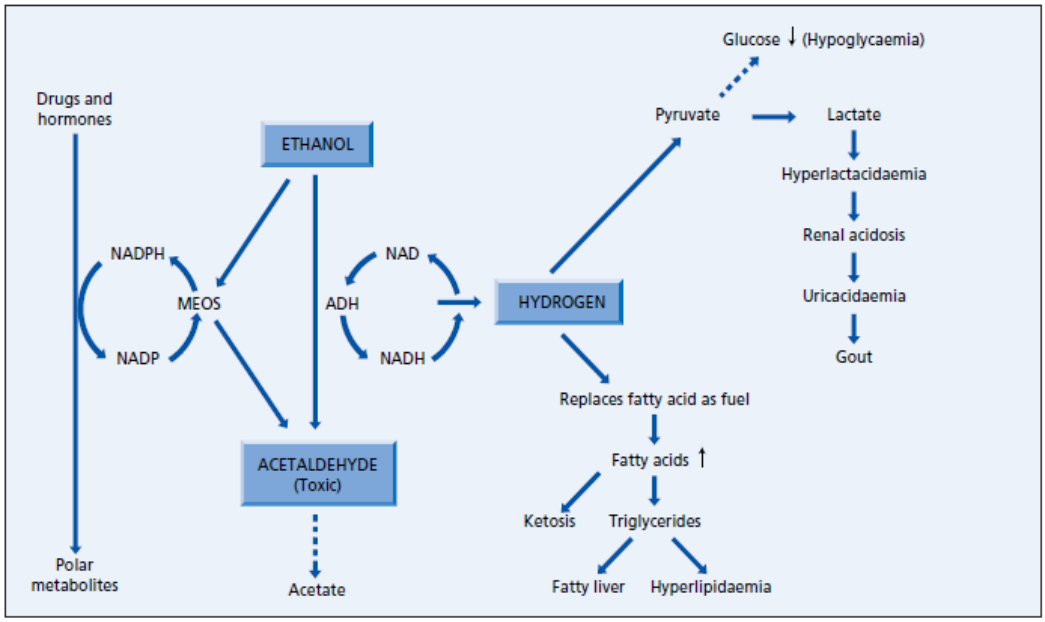
C	Have you felt the need to cut down?
A	Annoyed at the suggestion of a drinking problem
G	Guilty of excess drinking
E	Drink (eye opener) in the morning

METABOLISM OF ALCOHOL

80 percent of ethyl alcohol oxidized by alcohol dehydrogenale to acetaldehyde in hepatocyte cytoplasm.Normal liverwill metabolize 160-180 g of alohol per day .There will be enchanced production of acetaldehyhyde and its conversion to acetic acid is reduced. Acetaldehyde accumulates and causes damage to the cell membrane and Necrosis of the cell.

Figure: 3

ALCOHOL METABOLISM:



Hydrogen ion produced during acetaldehyde formation, replaces fatty acid as fuel and fatty aciod accumulation results in ketosis. Increased trigly cerides leads to fatty liver and hyperlipidemia.

Unwanted hydrogen used in conversion of pyruvate to lactase, results in renal acidosis, hyperuricemia and Gout. Reduction in pyruvate to glucose pathway leads to hypoglycemia.

GENETICS

Alcoholism patterns are inherited. Genetic polymorphisms in alcohol dehydrogenase and Aldehyde dehydrogenase are responsible for liver injury in some, but not in others, even they took same amount of alcohol for same duration[10].

Different rates of elimination is due to enzyme polymorphism.20% of alcohol oxidation takes place through CYP2E1[10]. There is no association between CYP2E1 polymorphism and liver injury[10].

Table: 5

RISK FACTORS FOR ALCOHOLIC LIVER DISEASE:

RISK FACTOR	COMMENT
1.Quantity	In men, 40-80 gm/day of ethanol produces fatty liver 160 g/day for 10-20 years causes Hepatitis or Cirrhosis. Only 15% of alcoholics develop alcoholic liver disease.
2. Gender	Women exhibit increased susceptibility to alcoholic Liver Disease at amounts >20g/day. Two drinks per day is probably safe.
3. Hepatitis C	HCV infection concurrent with alcoholic Liver Disease is associated with younger age for severity, more advanced histology and decreased survival.
4. Genetics	Patatin like phospholipase domain – containing protein 3 (PNPLA3) has been associated with alcoholic cirrhosis.
5. Fatty Liver	Alcohol Liver does not require malnutrition, but obesity and Non-alcoholic fatty Liver are risk factors. Patient should receive vigorous attention to nutritional support.

PATHOGENESIS

HEPATOTOXIC EFFECTS OF ACETALDEHYDE[11,12]

Acetaldehyde is highly toxic to liver and plays an important role in alcoholic hepatitis. Acetaldehyde forms adducts with protein or small molecules. These newer molecules alter the biologic process in the cell and injurious to it. These newer molecule induce host immune system and cause auto immune disease.

Glutathione transport in mitochondria is affected and leads to killing of hepatocytes by TNF mediated. Alteration in redox state of the cell affects the carbohydrate and lipid metabolism and decreased ATP supply to the cell, finally leads to fatty liver[11,12].

Table: 6

EFFECT OF ACETALDEHYDE ON LIVER

Increases lipid peroxidation
Binds plasma membranes
Interferes with mitochondrial electron-transport chain
Inhibits nuclear repair
Interferes with microtubule function
Forms adducts with proteins
Activates complement
Stimulates superoxide formation by neutrophils
Increases collagen synthesis

OXIDATIVE STRESS

Imbalance between pro and anti-oxidant. Over production of ROS (or) RNS and low level of antioxidants play a role and leads to hepatocyte injury through 2 pathways

1. Direct –lipid peroxidation and DNA damage
2. Indirect –signalling pathway transcription faster NFKB activation leads to production of TNF

MITOCHONDRIAL DYSFUNCTION

During acetaldehyde formation NADH produced and increases the redox state of the cell and oxidative stress. Increased superoxide generation occurs. To protect from oxidative stress mitochondria needs glutathione from cytoplasm. Transfer of glutathione is impaired and depletion of glutathione in mitochondria sensitize the liver cell to TNF toxic effect and apoptosis occurs[11,12].

HYPOXIA

In hepatic lobule centrilubuar area is more prone for hypoxia due to low oxygen tension. Depletion in ATP occurs, reduced state of the cell increased, free radical generation and elevated liver enzymes occurs[11,12].

IMPAIRED PROTEASOME FUNCTION

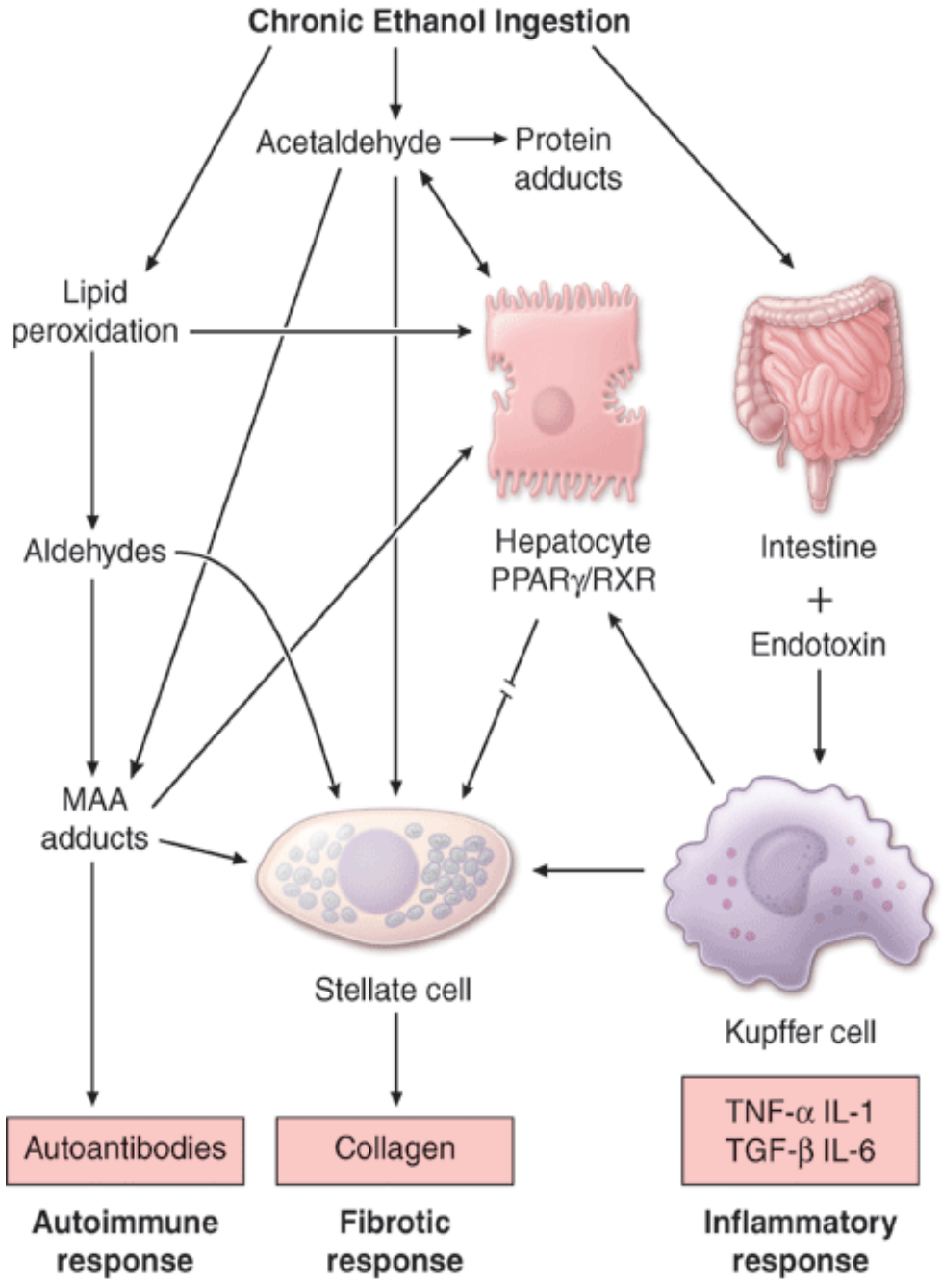
Proteolytic activity decreased in proteasome which leads to accumulation of protein (oxidized) serum ubiquitin level will be increased.IL-8 and IL-18 released, causes neutrophil recruitment and inflammation respectively[11,12].

ABNORMALITY IN METHIONINE, FOLATE SAM METABOLISM

Activity of the enzyme methionine adenosyl transferase is inhibited, which further affects the conversion of methionine to SAM. Homocysteine and SAH will be increased .SAM/SAH ratio is decreased and inhibits the trans methylation reaction of DNA, RNA, biogenic amines, histones, phospholipids and others [11,12].

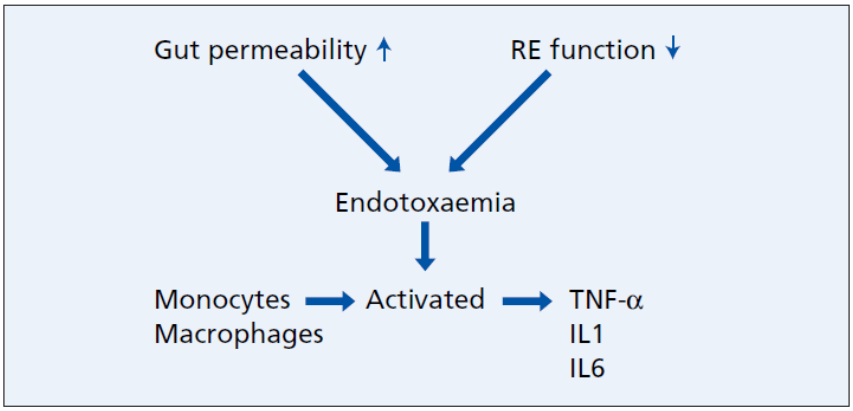
Due to SAM deficiency, glutathione is depleted. SAM decreases Lipopoly Saccharide mediated TNF release and increases the cytokine IL-10, depletion of SAM in alcoholics has reverse effect. SAH mediates TNF effect on hepatocytes and causes injury. Folate deficiency prevents the conversion of Homocystine to Methionine and aggravates liver injury in alcoholic with folate deficiency

Figure: 4 Pathogenesis of Alcoholic Liver disease



INFLAMMATARY AND IMMUNE MECHANISMS[11,12]

ROS and LPS derived from the gut activates the kupffer cells and NFKB transcription factor results in release of TNF and proinflammatory cytokines.

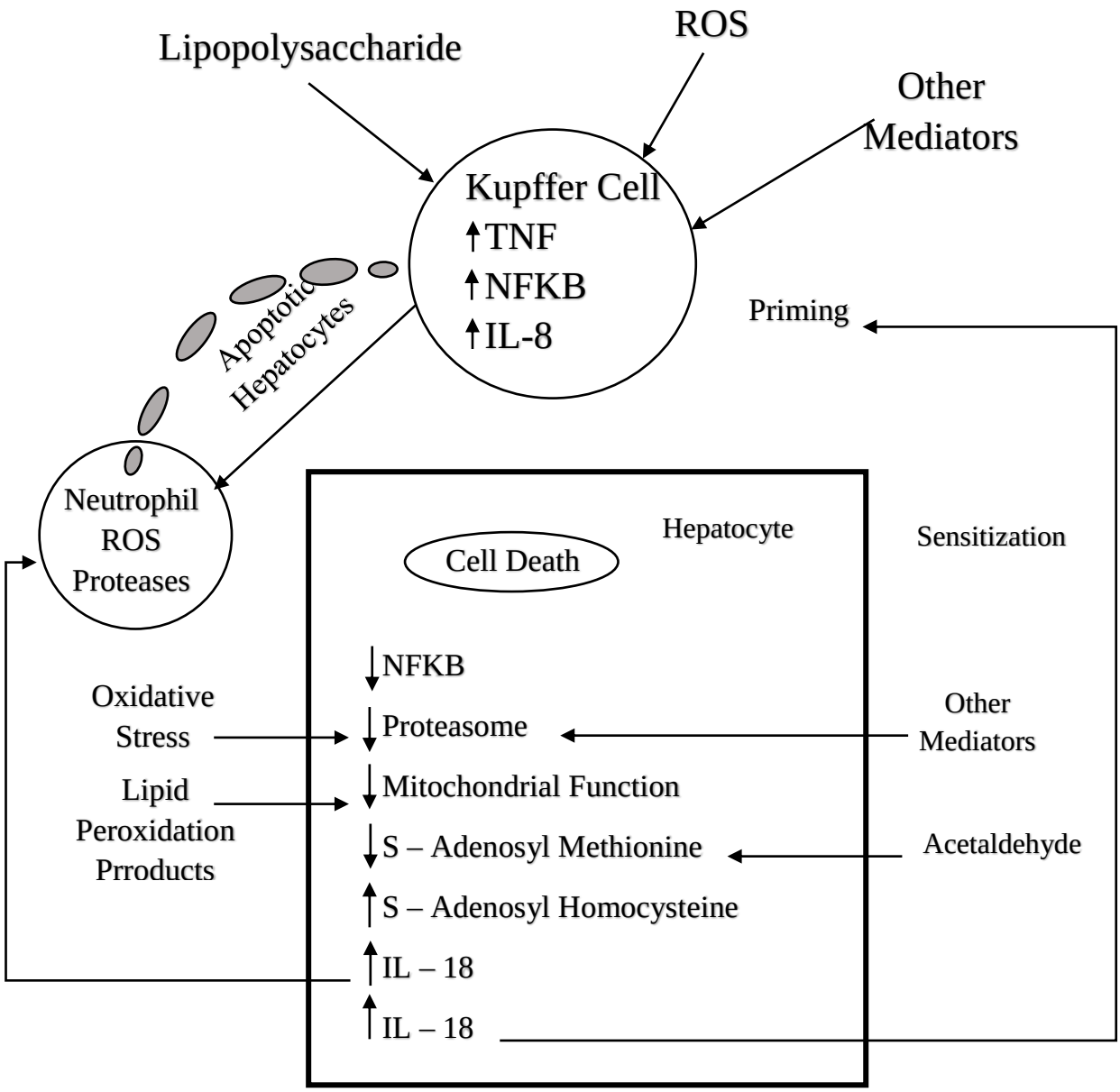


IL-8 and IL-18 released from dying hepatocytes cause inflammation. Both necrosis apoptosis occurs.

TGF-B activates stellate cells to produce collagen and result in fibrosis. Anti-inflammatory cytokines IL -10 will be decreased[11,12].

Interaction of alcohol metabolites with liver proteins results in the formation of new antigens. Host immune response to this new antigen results in auto immune disease[11,12].

Figure: 5 Inflammatory and Immune Mechanism

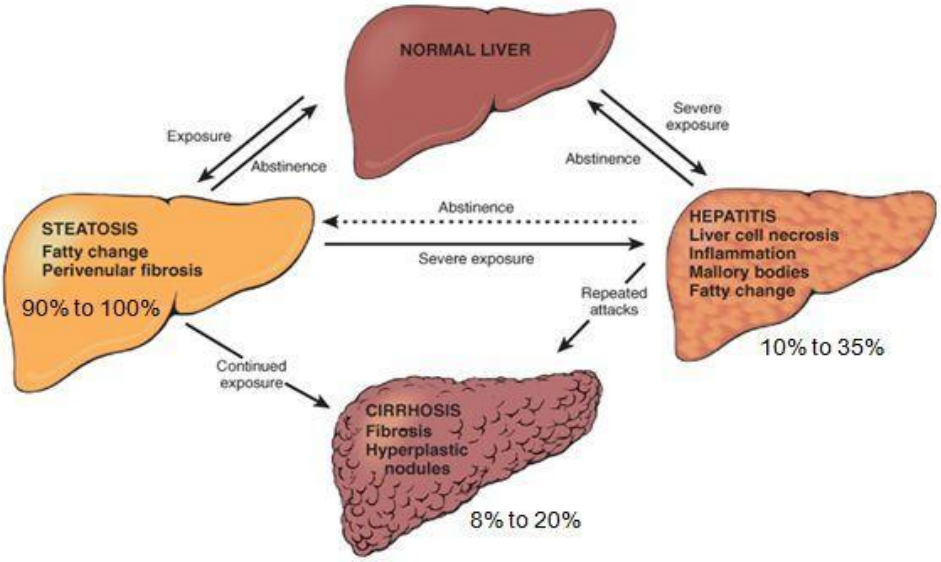


MORPHOLOGY

Morphological changes are classified in to[13]

1. Fatty liver
2. Alcoholic hepatitis
3. Cirrhosis

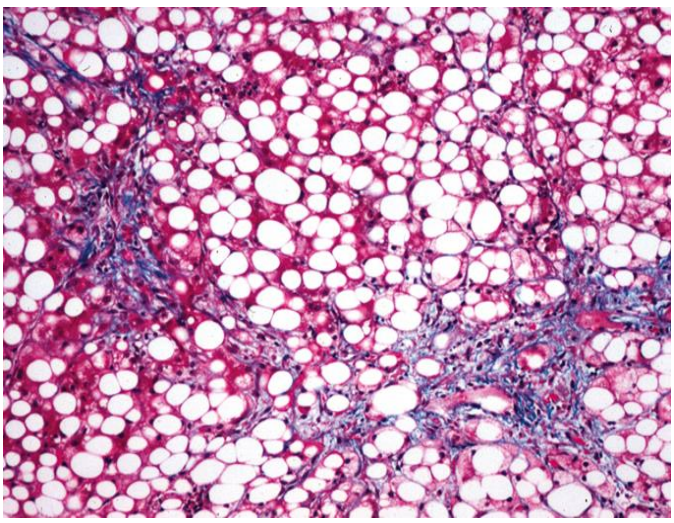
Figure: 6 Spectrum and Interrelationship of ALD



FATTY LIVER

The fatty changes are micro vesicular initially with moderate consumption of alcohol. With chronic alcohol intake macro vesicular changes are seen[13]. Fatty change will be centrilubulor initially and later involves the entire lobule. Even in binge drinking, fatty liver develops within 3-7 days. The zones 3 and 2 are accumulated by fat[13].

Figure: 7 Histopathology of Fatty Liver



QUANTIFICATION OF FATTY CHANGE

1 + < 25% of hepatocytes contains fat

2 + 25-50% of hepatocytes contains fat

3 + 50-75 % of hepatocytes contains fat

4 + >75% of hepatocytes contains fat

ALCOHOLIC HEPATITIS

It may be seen separately or occurs in a patient with established Cirrhosis.

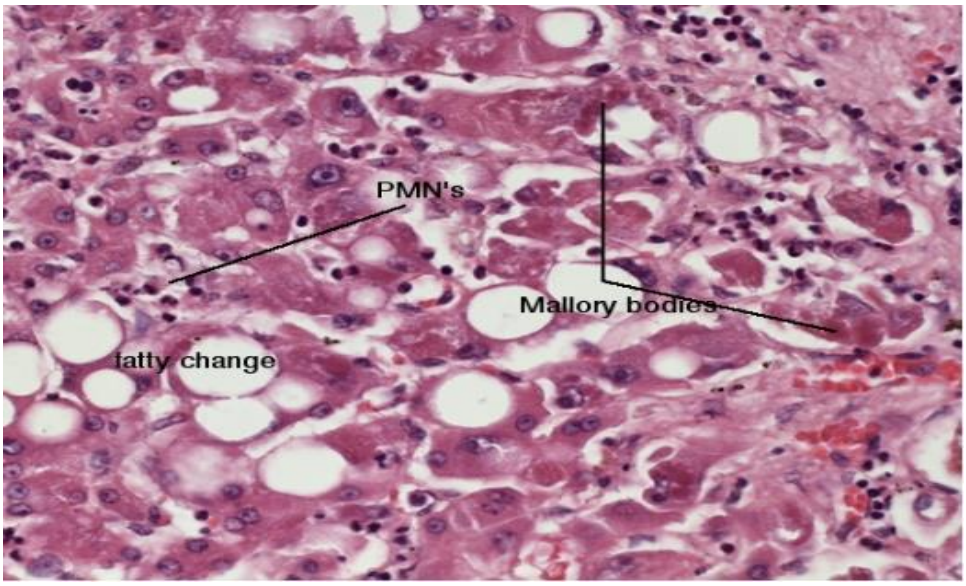
1. **HEPATOCYTE SWELLING AND NECROSIS**

   Ballooning of Hepatocytes due to water retention and failure of protein excretion by the microtubules. Fatty change is usually macro vesicular. Swelled Hepatocyte will cause Cholestasis[13].

2. **MALLORY BODIES**

   Cytokeratin intermediate filaments accumulate in degenerating Hepatocytes as Cytoplasmic inclusions[13]. These are characteristic of alcoholic Hepatitis but not specific Mallory bodies target the Liver cell for destruction.

Figure: 8 Histopathology of Alcoholic Hepatitis



3. NEUTROPHILIC REACTION

Neutrophils get accumulated around Mallory bodies in degenerating Hepatocytes[13].

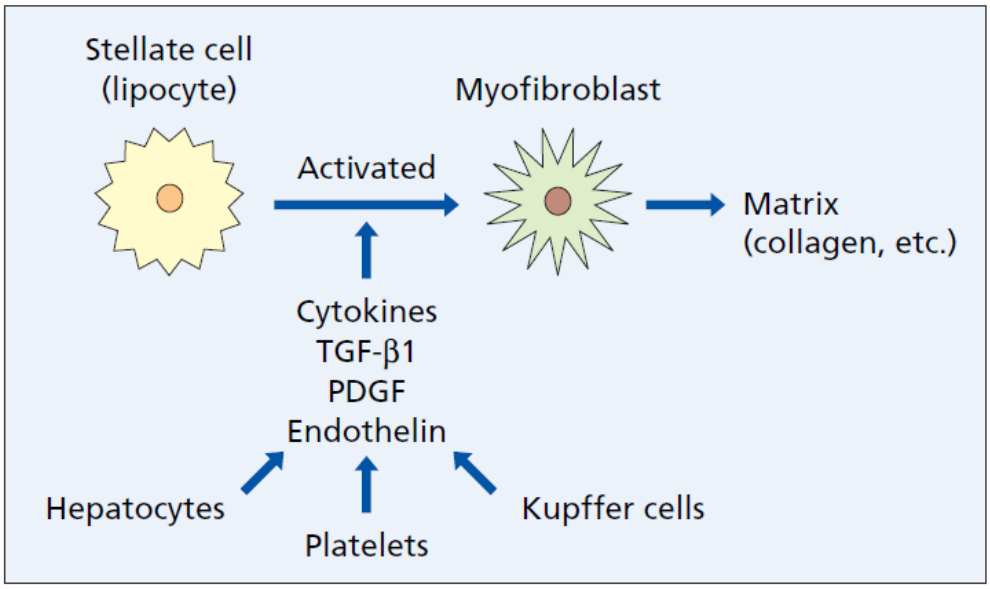
4. FIBROSIS

Sinusoidal stellate cells are activated by Cytokines TGF-β, PDGF, Endothelia and activation of portal tract fibroblasts leads to fibrosis[13]. Fibrosis is in sinusoidal and perivenular area leading to portal Hypertension[13].

Maximal collagen deposition is in zone 3. Collagen also deposit in space of dissce. Alcoholic Hepatitis predispose to Cirrhosis[13].

Figure: 9

Stellate Cell Activation

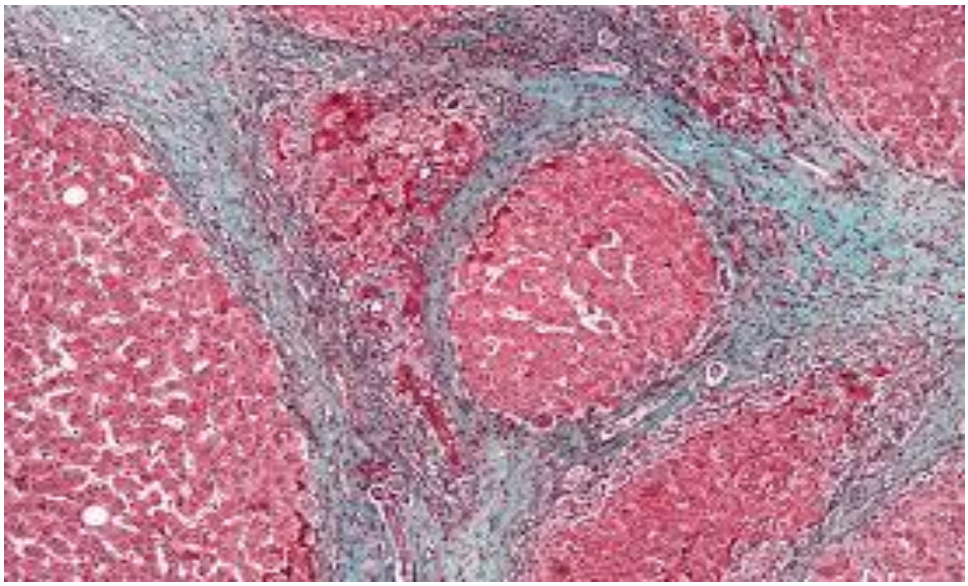


5. CIRRHOSIS

Alcoholic Cirrhosis is usually micro nodular. Changed to macro nodular due to continued necrosis and fibrosis, accompanied by reduced Steatosis. The zonal architecture are difficult to identify. In this end stage, it is difficult to find the etiology on histological examination because of similarity to viral hepatitis[13].

In one third of patients Hepatic Iron will be increased. Ischemic Necrosis and Nodular Fibrous obliteration leads to tough pale scar tissue. (Laennec Cirrhosis).

Figure: 10 Histopathology of Cirrhosis Liver



EFFECTS OF ALCOHOL ON OTHER SYSTEM

NERVOUS SYSTEM:

1. Peripheral Neuropathy in 10% of Alcoholics.
2. Cerebellar Degeneration or Cerebellar atrophy is 1%
3. Wernicke's encephalopathy (Ophthalmoplegia, Ataxia and Encephalopathy)
4. Korsakoff's syndrome (Retrograde and Anterograde Amnesia)
5. Cognitive problems.

PSYCHIATRIC COMORBIDITY

1. Anxiety, Sadness
2. Alcohol induced Psychotic disorder.
3. Hallucination

CARDIAVASCULAR SYSTEM

1. Atrial or Ventricular Arrhythmias – Paroxysmal tachycardia
2. Cardiomyopathy
3. Hypertension / Heart failure

OTHERS

- Testicular Atrophy
- Fetal Alcohol Syndrome
- Alcoholic Myopathy
- Increased risk of fractures
- Femoral Head Osteonecrosis
- Elevated Cortisol Level
- Decrease in Thyroid Hormones

HAEMATOLOGICAL MANIFESTATIONS IN CHRONIC ALCOHOL CONSUMERS

1) Alcohol has both direct and indirect adverse effects on the hematological system. Direct adverse effects includes toxicity of Ethanol or Acetaldehyde on bone marrow, precursors of blood cells and mature RBC's, WBC's and Platelets.

Indirect adverse effects includes Nutritional deficiencies like Iron deficiency and Folic acid deficiency.

2) Toxic effects of alcohol are dose dependent, so significant impairment in Hematopoiesis occurs in severe Alcoholics only.
3) In chronic severe alcoholics the precursors of blood cells in the bone marrow are reduced and abnormality in the cell structure leads to decreased functional mature blood cells.
4) Binge drinking of alcohol may results in acute suppression of bone marrow and results in Thrombocytopenia, decreased reticulocyte and Granulocyte levels.
5) Nutritional factors like Iron deficiency sometimes associated with Folic acid deficiency in alcoholics leads to Dimorphic Anemia.
6) Hence, alcoholics may present with moderate anemia, which encompasses by macrocytic RBC's and abnormal RBC's structure, mildly reduced WBC's numbers especially Neutrophils and moderate to severe Thrombocytopenia. Pancytopenia may be seen sometimes and it is not progressive usually and reversed with abstinence.

VACUOLES DEVELOPMENT IN RBC PRECURSORS:

- Vacuolation of precursors occurs in either erythroid or myeloid series. It is reversible.
- The colony forming unit of early (BFU erythroid) and late (CFU erythroid) of human erythroid progenitor cells are suppressed more by Ethanol / Acetaldehyde, When compared with myeloid cell progenitors.
- Vacuoles in precursor cells of RBC is the most characteristic finding in bone marrow and it indicates the direct toxicity of alcohol on bone marrow.
- Alterations in the RBC membrane structure plays an important role in vacuole formation and it indicates alcohol consumption in excess.
- Vacuoles appear in the early red cell precursors, after 5-7 days of excess alcohol intake and disappears by 3-7 days after abstinence. Sometimes they may persist for up to 2 weeks.

SIDEROBLASTIC ANEMIA:

- 25-30% of anaemic alcoholic patients will have ring sideroblasts in bone marrow. Sideroblastic anemia in alcoholics may be due to interference with the enzyme activity, involved in heme synthesis.

- Decreased activity of enzymes like erythrocyte ALA dehydratase (due to zinc depletion), uroporphyrinogen decarboxylase, coproporphyrinogen oxidase and Ferrochelatase. There will be increased activity of ALA synthase and PBG deaminase. Finally results in impaired heme production.
- Iron is not incorporated into the Haemoglobin molecule properly and it is converted to Ferritin, Storage form, which accumulates in precursors of RBC's. As granules around the cell's nucleus. These cells are called as ringed sideroblasts.
- These cells cannot mature into functional RBC's so RBC number in blood declines and results in Anemia. Many patients have some circulating RBC's that contain Ferritin granules called as pappenheimer bodies. Sideroblastic Anemia is a common complication of severe alcoholics.
- The percentage of ring sideroblast in a bone marrow ranges from 10-70%. Abstinence from alcohol reverse this changes from the bone marrow with in few days to 2 weeks.

EFFECT OF ALCOHOL ON IRON METABOLISM

- Alcohol will enhances iron absorption from the gastrointestinal tract. So marrow iron stores will be increased, serum Ferritin and Transferrin saturation also will be increased.

- Iron deficiency in alcoholics may be due to bleeding from the gastrointestinal tract. So alcohol can results in either iron deficiency or iron excess in the body.
- Iron deficiency is difficult to diagnose in alcoholics due to masking of symptoms of Folic acid deficiency or coexisting liver disease and other alcohol related inflammatory conditions.
- Increased iron lever sometimes leads to hemochromatosis. Hemochromatosis complicates to Cirrhosis liver and Hepatocellular Carcinoma.

MEGALOBLASTIC ANEMIA:

★ In alcoholics megaloblastic anemia is usually due to folic acid deficiency.

★ Direct toxic effect of alcohol on bone marrow → macrocytosis.

★ Folate deficiency → megaloblastic anemia.

★ Alcoholic cirrhosis → spur cell hemolytic anemia.

★ Alcohol will acutely depress the serum Folic acid, even if stores of folate are full.

★ In some patients with early folate deficiency, alcohol will accelerate the development of megaloblastic anemia. These changes will occur even if large doses of folate are given with alcohol.

- ★ Anemia in alcoholics with megaloblastic bone marrow changes are always due to Folate deficiency megaloblastic anemia will be rarely seen in non-hospitalized chronic alcoholics or relatively well nourished subjects, admitted in hospital for alcohol withdrawal.
- ★ Alcohol ingestion decreases the Folate absorption from food.
- ★ Due to folate deficiency, precursor cells cannot divide properly and results in accumulation of large immature and Non-fucntional cells megaloblasts in bone marrow and blood stream. This impaired Hematopoiesis mainly affects RBC's, WBC's and Platelets also involved.

RBC DISORDERS IN ALCOHOLICS:

- ★ The morphological abnormality, Macrocytosis, may be an initial findings followed by target cells and acanthocytes.
- ★ In RBC membrane, there will be abnormal cholesterol loading. Integral membrane protein mobility is restricted due to abnormal cholesterol loading of the Lipid bi layer. This restricts the normal deformation of the erythrocytes while passing through the micro vasculature.
- ★ In case of severe cholesterol loading, when erythrocytes passing through microcirculation of spleen, cytoskeletal damage to RBC

membrane occurs and irreversible deformity noted in RBC morphologically as spur cells.

- ★ Since liver is integrally involved in lipid metabolism and lipids are major constituents of RBC membrane. Size and shape abnormalities in erythrocytes may arise with hepatic Dysfuntion.
- ★ Structurally abnormal RBC's undergoes premature or accelerated destruction (hemolysis) and presents with Anemia.
- ★ Presence of Ovalocytes and elliptocytosis in peripheral smear is due to Iron deficiency.
- ★ Acanthocytes is due to congestive splenomegaly, toxic effect of alcohol and liver disease. Stomatocytes is seen in alcoholics.
- ★ Schistocytes are due to hemolysis.
- ★ Presence of target cells indicates iron deficiency.
- ★ Tear drop cells in alcoholics are due to splenomegaly in decompensated liver disease.

MACROCYTOSIS:

- ✓ Degree of alcoholic macrocytosis is mild and MCV ranges from 100-110 fl. 82-96% of chronic alcoholics had macrocytosis, unrelated to presence of liver disease / Folic acid deficiency.
- ✓ Macrocytosis is due to direct toxic effect of alcohol on bone marrow and it is a screening procedure for detection of alcoholism.

- ✓ Macrocytosis is common in alcoholics, but it does not always indicate the megaloblastic Anemia.
- ✓ The enlarged RBC's are round in case of alcoholic macrocytosis, in contract to oval shaped in megaloblastic Anemia. This helps to differentiate both.
- ✓ MCV comes to normal with complete abstinence of alcohol in 2-4 months.

CAUSES OF ANEMIA IN ALCOHOLICS

In case of chronic alcohol ingestion, Anemia is due to

1) Nutritional Deficiency - Iron and Folic acid.
2) Chronic G.I. Bleeding - Iron deficiency.
3) Direct toxic effect of alcohol on erythropoiesis.
4) Hepatic Dysfunction.
5) Hyper splenism.

Folate deficiency results from decreased intake, decreased absorption and abnormal metabolism[21].

- 82% of men and 44% of women with macrocytosis are found to be alcoholics.
- Macrocytosis develops in alcoholics even in the absence of liver disease or Folate deficiency. Gastro intestinal blood loss occur due to peptic ulcer or thrombocytopenia.

HEMOLYTIC ANEMIA

Causes of Hemolysis in alcoholism

1) Hypophosphatemia
2) Presence of malformed RBC's – stomatocytes and spur cells.

- ✓ More than 25 percent of alcoholics have stomatocytosis. Alcoholic liver disease also plays a role in stomatocyte hemolysis. In stomatocytes the RBC membrane defect causes the cells to an abnormal stoma like shape, have short life span due to entrapment in the small capillaries of spleen and destroyed. Spur cell Hemolysis – Spur cells are RBC's with spike like protusions from the cell membrane caused by severe cholesterol loading in to the cell membrane.
- ✓ These cells have increase in cell surface area with a corresponding increasing in cell volume. While passing through the splenic micro capillaries these cells undergoes irreversible cytoskeletal damage and prematurely destroyed.
- ✓ 3% of alcoholics with advanced liver disease have spur cell hemolysis. Spur cell hemolysis in patient with liver disease indicates poor prognosis.
- ✓ Hypophosphatemia – Hemolysis due to hypophosphatemia is rare. Alcohol causes hypophosphatemia due to increased urinary excretion of phosphates, Seen commonly in withdrawal phase.

Phosphate and ATP levels in RBC decreased and leads to hyper rigidity of the RBC membrane and damages the cells. These membrane damaged cells finally destroyed in the spleen, leads to Acute Hemolytic Anemia.

EFFECTS OF ALCOHOL ON WBC's

Due to direct toxic effect of alcohol, Neutrophil development from the precursor cells impaired and results in infections like Bacterial pneumonia, Lung abscess or other Bacterial Infections, much more common, When compared with Non-alcoholics.

Alcohol also impairs the monocytes and macrophages function and Lymphocytes also affected.

In case of alcoholics with Bacterial Infection, initially transient Neutropenia will be present, then rebound Lenkocytosis occurred between 5 and 10 days after the infection. Neutropenia is due to impaired Neutrophil development from the precursors. The quick response of Neutrophil stores in the bone marrow to a bacterial infection is depleted more rapidly in alcoholics, when compared with Non-alcoholics. Neutrophil delivery to the infection / inflammation site also affected.

Alcohol interfere with Leukotriene production, which regulates Neutrophil function. Reduced Leukotriene production and Neutrophil's inability to respond to Leukotriene explain the Neutrophil dysfunction in alcoholics.

MANOCYTES AND MACROPHAGES

Alcohol inhibits the adhesion abilities of the monocyte and macrophages. Mainly the alteration in function, rather than the number of monocytes.

EFFECT OF ALCOHOL ON THE BLOOD CLOTTING SYSTEM

Alcohol interferes with coagulation cascade at several Levels and affects the fibrin formation. Fibrinolysis also affected results in the formation of thrombus and leads to stroke.

THROMBOCYTOPENIA

Thrombocytopenia is frequently seen in alcoholism. Affects 13 to 43% of non-acutely ill, well-nourished alcoholics and 16 to 82% of acutely ill hospitalized alcoholics.

Thrombocytopenia is due to

1) Decreased platelet survival
2) Decreased platelet turnover

There will be accelerated platelet destruction and subnormal compensatory increase in Thrombopoiesis. Alcohol induced thrombocytopenia is transient and within 2-3 days platelet count come to normal or supernormal within 2-3 weeks after abstinence[5].

Usually the patients do not have bleeding manifestations and no therapeutic intervention needed expect for most severe cases. Rebound

thrombocytosis occurs in most of the patients after 2 to 3 weeks of alcohol cessation.

Alcohol Induced Thrombocytopenia is due to direct toxic effect of alcohol. Interfere with the late stage of platelet production and also life span of the remaining platelets also shortened. In chronic alcoholics, Thrombocytopenia is due to Liver Cirrhosis, Congestive Splenomegaly and Folic acid deficiency. Acute thrombocytopenia is due to direct bone marrow suppression by alcohol.

THROMBOCYTOPATHY

Not only platelet production, but also platelet function also affected by alcohol. Impaired platelet aggregation, decreased secretion or activity of clotting factors. Vitamin K dependent clotting factors II, VII, IX, X are affected because alcohol impairs the clotting mechanism. Interaction with certain drugs like Aspirin or other NSAIDS, further prolongs the bleeding time. Alcohol also interact with anticoagulants warfarin.

FIBRINOLYSIS

Finrinolytic activity will be decreased in alcoholics and they will prone for thrombosis.

- ✓ Clotting factor synthesis will be decreased

- ✓ In liver disease due to abnormal post translational, modification, defective form of fibrinogen will be synthezised.
- ✓ These defective fibrin molecules are not able to polymerize into fibrin strands. Thrombin time and reptilase time (measures of fibrinogen polymerization into fibrin) are prolonged, often in the setting of normal Immunologic amount of fibrinogen. Because fibrin is a polymer of many subunits, even small number of abnormal fibrin molecules can poison the multimerization process and results in bleeding diathesis.

HEMATOLOGICAL MARKERS OF ALCOHOLISM

1) State markers – For identification of heavy drinkers.
    a) Carbohydrate Deficient Transferrin.
    b) Mean corpuscular volume.
2) Trait markers – Identifies people at risk for alcoholism
    a) Mono amine oxidase.
    b) Adenylyl cyclase.

VITAMIN B_{12} AND FOLIC ACID

Folate is an important cofactor in thymidylate synthesis, which in turn is essential for DNA synthesis. Synthesis of RNA is not affected, so

Folate deficiency results in Nuclear Cytoplasmic dissociation, which is characteristic of Megaloblastic Anemia.

Cobalamin is essential for synthesis of S-adenosyl Methionine, which in turn used in various neurotransmitters synthesis. So its deficiency results in Neurological manifestations, corrected by giving Cobalamin and not by Folate.

Cobalamin helps in Folate recycling. Vitamin B_{12} deficiency causes Hematological changes by restricting the supply of Folate. So Hematological changes due to Vitamin B_{12} deficit can be corrected by giving excess Folate. But Folate deficiency Hematological changes cannot be corrected with vitamin B_{12}.

Megaloblastic Anemia is due to vitamin B_{12} or Folic acid caused by impaired DNA synthesis.

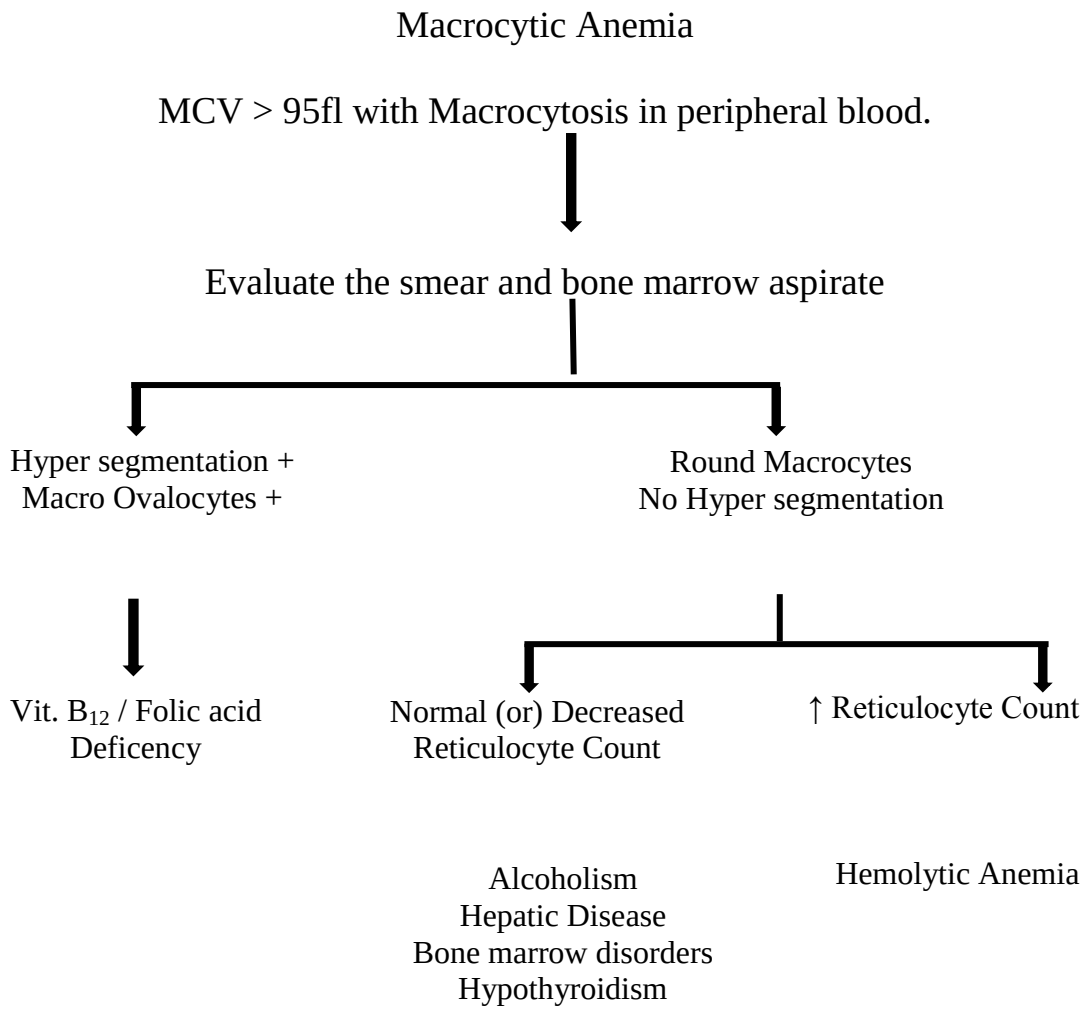
Gives rise to Macro Ovalocytic red cells with intense Erythroid hyperplasia and Megaloblastic changes in the bone marrow.

Alcohol abuse results in sharp fall in serum Folate within 2-4 days by impairing it's entero hepatic cycle and inhibiting its absorption.

Sr. Folic acid – 5.4 to 18 ng / ml.

Sr. Vitamin B_{12} – 279 to 966 pg / ml.

High level of serum. Cobalamin is seen in liver disease like Cirrhosis, Secondaries Liver, Carcinoma Liver and acute alcoholic hepatitis due to Cobalamin release during Hepatocyte. Injury and decreased clearance by the Liver.



Elevated serum vitamin B_{12} is seen in acute Leukemia, Polycythemia Vera, Hypereosinophilic syndrome due to enhanced production of the transport protein Haptocorrin.

PROTHROMBIN TIME

In case of Chronic Alcohol abuse, Prothrombin time will be usually elevated. This is due to deficient in the synthesis of clotting factors such as II, VII, V, X. it is used to assess the prognosis in acute liver failure. If it is elevated, the prognosis will be poor.

In chronic alcoholic Liver disease, Serum albumin level is used to monitor the disease progression. Serum albumin level will be decreased and globulin level will be increased and there is reversal of Albumin globulin ratio. Increased globulin is due to more antibody formation against the intestinal bacterial antigen, as it is not cleared by the Liver. Alcohol intermediate form a newer molecule with Liver protein and host immune response against this newer antigen also results in increased level of globulin.

The ratio of SGOT and SGPT will be elevated in chronic alcohol abusers. If the ratio is more than 3, it is highly suggestive of liver disease due to alcohol etiology. Usually, SGOT and SGPT will be less than 300. If it is more than 300, alternative diagnosis should be made. GGT is neither sensitive nor specific for the diagnosis. Macrocytosis along with elevated GGT have a higher sensitivity. Leukocytosis and elevated SGOT more than 300 is commonly seen in acute alcoholic Hepatitis and it is difficult to differentiate from associated infections.

TYPICAL LABORATORY ABNORMALITIES IN ALCOHOLIC LIVER DISEASE

LIVER FUNCTION TESTS:

- AST > ALT (SGOT > SGPT)
- AST usually < 300 IU and ALT <200 IU
- Serum Albumin decreased

- Elevated PT, Serum Bilirubin
- Elevated GGT, Globulins, Alkaline phosphatase

HAEMATOLOGIC ABNORMALITIES:

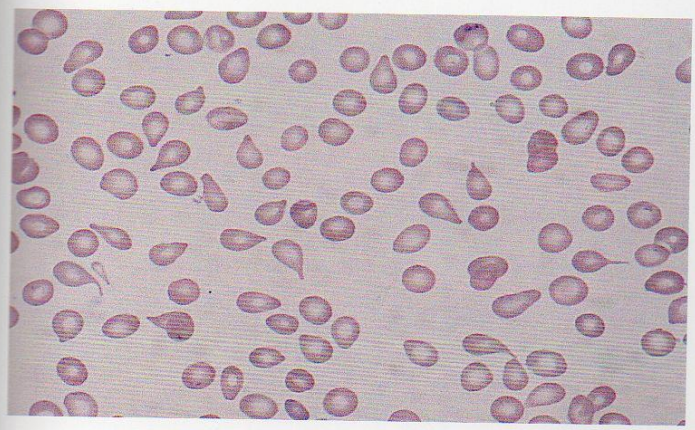
- Microcytic, Hypochromic (or) Macrocytosis (or) Dimorphic Blood Picture (or) Pancytopenia.
- Thrombocytopenia, Decreased MCH, MCHC.
- Elevated white blood cell count especially Neutrophils.
- Leukaemoid reactions associated with alcoholic hepatitis.
- Stomatocytes, spur cells, target cells in peripheral smear.
- Sehistocytes, tear drop cells, elliptocytes, Ovalocytes in peripheral smear.
- Decreased Folic acid with normal or elevated B_{12} level.
- Bone marrow – vacuoles in RBC precursors, sideroblastic Anemia, Megaloblastic changes.

METABOLIC ALTERATIONS:

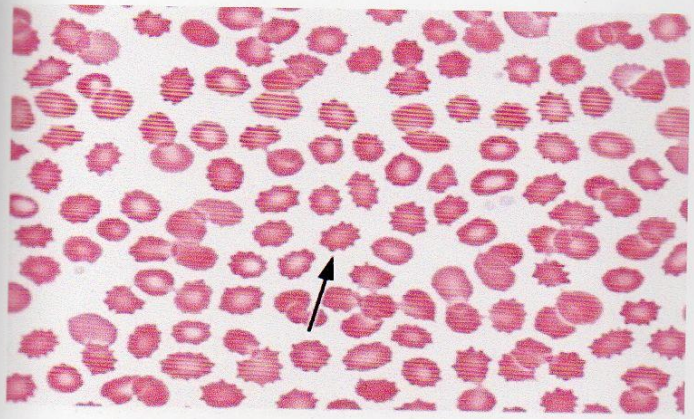
- Hypertrigly ceridaemia
- Hypernatremia, hypokalemia, hypophosphatemia, hypomagnesemia.
- Hyperglycemia / Hypoglycemia

Abnormal RBC Morphology in Alcoholics

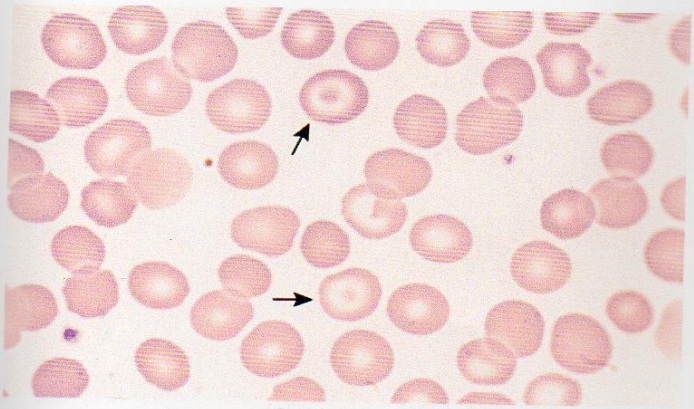
igure: 11 Tear Drop Cells



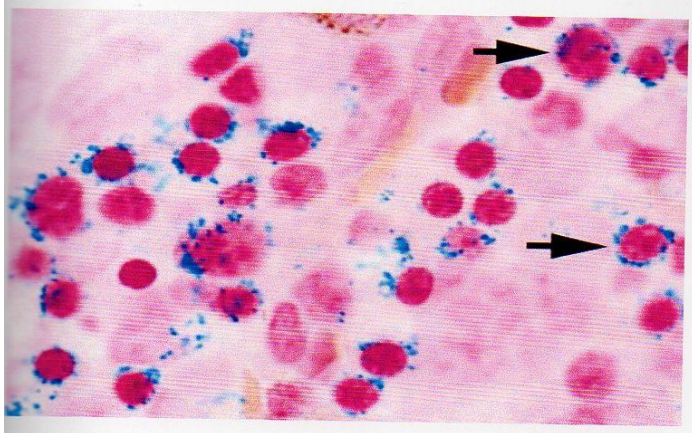
gure: 12 Spur Cells



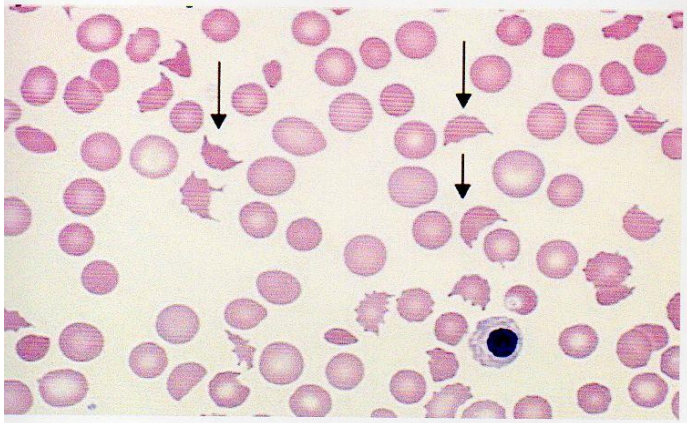
gure: 13 Macrocytosis with Target Cells



gure: 14 Ring Sideroblast in Bone Marrow



e: 15 Schistocytes



e: 16 Stomatocytes

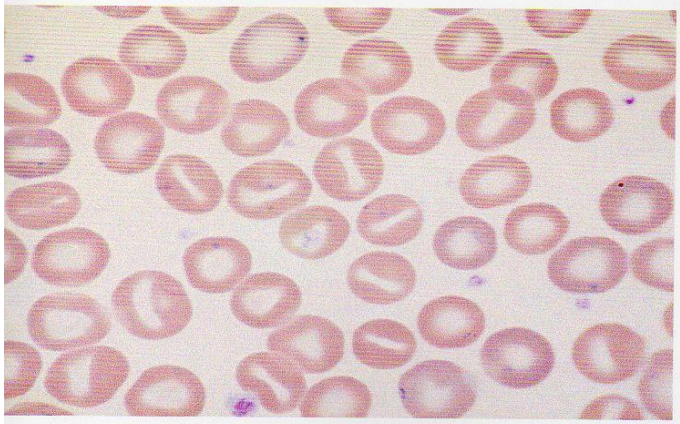


Figure: 17 Hypochromic Microcytic

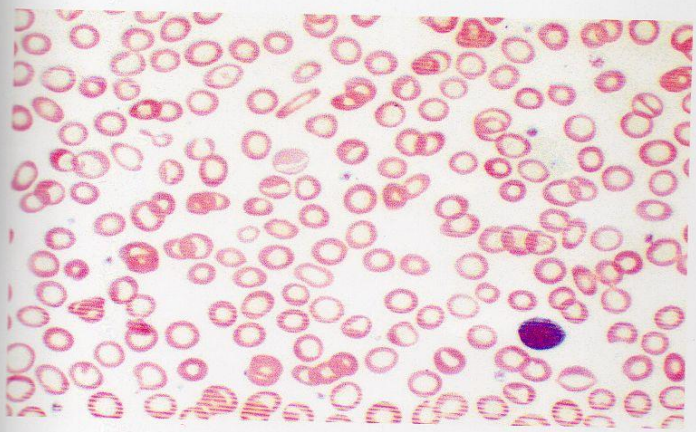


Figure: 18 Ovalocyte and Elliptocyte

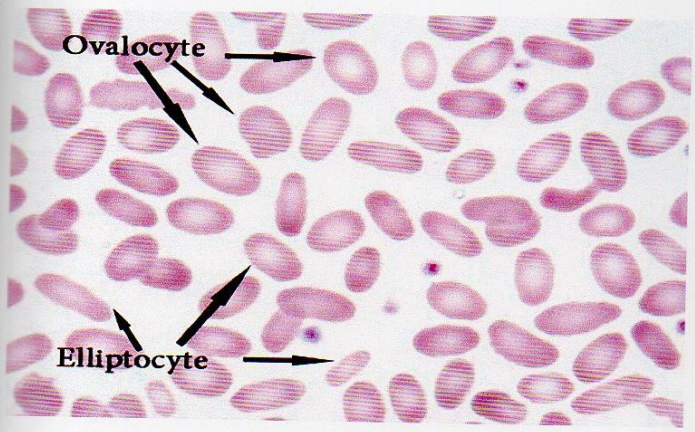


Figure: 19 Vacuolated RBC Precursors

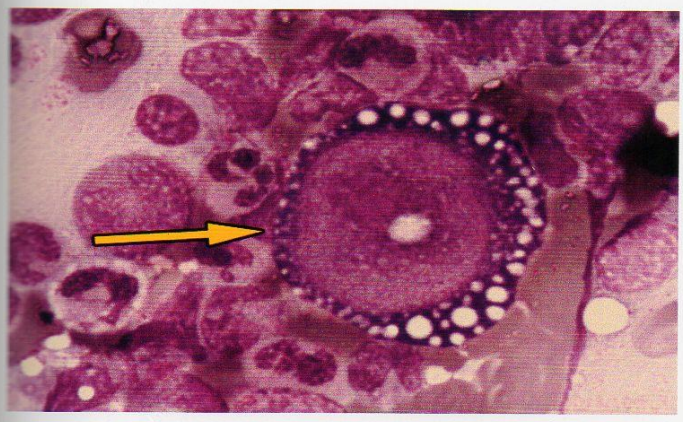
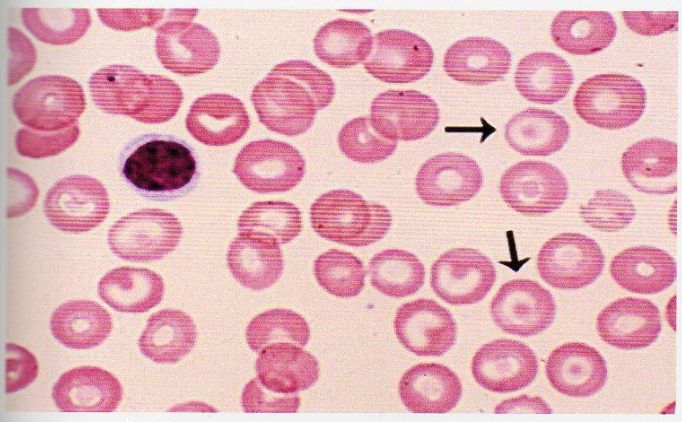


Figure: 20 Macrocytosis



Materials and Methods

MATERIALS AND METHODS

SETTING	:	Govt. Mohan Kumaramangalam Medical College Hospital, Salem.
COLLABORATIVE	:	Department of Pathology Department of Bio-chemistry Govt. Mohan Kumaramangalam Medical College Hospital, Salem.
STUDY DESIGN	:	Prospective Cross Sectional Study
STUDY PERIOD	:	August 2014 – August 2015.
SAMPLE SIZE	:	100 cases, 50 Alcoholics (25 moderate alcoholics, 25 severe alcoholics) and 50 non-alcoholics.
ETHICAL COMMITTEE APPROVAL	:	The Present Study was approved by the Ethical Committee.

INCLUSION CRITERIA

- All adult patients who are moderate alcoholics, defined as less than or equal to two drinkers per day for men and less than or equal to one drink per day for women.
- All adult patients who are severe alcoholics, defined as more than 7 drinks a week in women and more than 14 drinks in a week in men.
- 50 adult patients who are non-alcoholic taken as controls.
- Both sexes were included.

EXCLUSION CRITERIA

- Patients with age less than 18 years.
- Pregnancy
- Previous history of Haematological Malignancies.
- Patients with other hepatic diseases.
- Chronic illness such as Tuberculosis (or) Diabetes mellitus.
- Patients receiving any hepatotoxic drugs.

CONTROLS

50 non-alcoholics patients who met the above exclusion criteria.

CONSENT

With the above criteria the study group was selected and instructions given about the study. Written informed consent got from the willing participants.

MATERIALS

50 alcoholics (25 moderate and 25 heavy drinkers) and 50 non-alcoholics.

CONFLICT OF INTEREST

There was no conflict of interest.

FINANCIAL SUPPORT

Nil

METHODS

The following data's were collected from the cases and controls and recorded in the proforma.

1. Demographic data
    - Name
    - Age
    - Sex
    - Occupation
    - Socio-economic Status
2. History
    - Jaundice, Abdominal distension, Pedal oedema, Swelling of Legs, Haematemesis, Malena, Altered sensorium.
3. Alcohol Intake
    - Amount of alcohol intake, moderate or severe drinkers, duration.
4. Clinical Data
    a) General examination – Pallor, Icterus, Pedal oedema and Sign of Hepatic failure.
    b) System examination – per abdomen, Cardiac, Respiratory and Central nervous system.
5. Laboratory Data

a) Complete Blood Count (by Automated Counter)
   - ☻ Haemoglobin in gm%
   - ☻ RBC count in million / cmm
   - ☻ PCV in %
   - ☻ Total WBC count in cells / cmm
   - ☻ MCV in fl
   - ☻ MCH in pg
   - ☻ MCHC in %
   - ☻ Platelet count in lakhs/ cmm

b) Microscopic peripheral smear study.

c) Prothrombin time in seconds.

d) Serum vitamin B_{12} in pg/ml. (by chemiluminescent micro particles immuno assay)

e) Serum Folic Acid in ng/ml (by chemiluminescent immuno assay)

f) Liver function tests – Total Bilirubin, Direct and Indirect fraction, SGOT, SGPT, Total protein, Albumin and Alkaline phosphatase.

g) Renal function tests – Blood urea, Serum Creatinine

h) Random Blood Sugar, USG abdomen, echo cardiogram.

STATISTICAL ANALYSIS

Data of 100 patients was collected and entered in a Microsoft excel sheet.Statistical analysis done by using SPSS11.5 version.

Chi square tests was applied for comparing discrete variables and student ‘t’ tests was applied for comparing continuous variables ‘p’ value of <0.05 was significant statistically.

Results & Analysis

RESULTS AND ANALYSIS

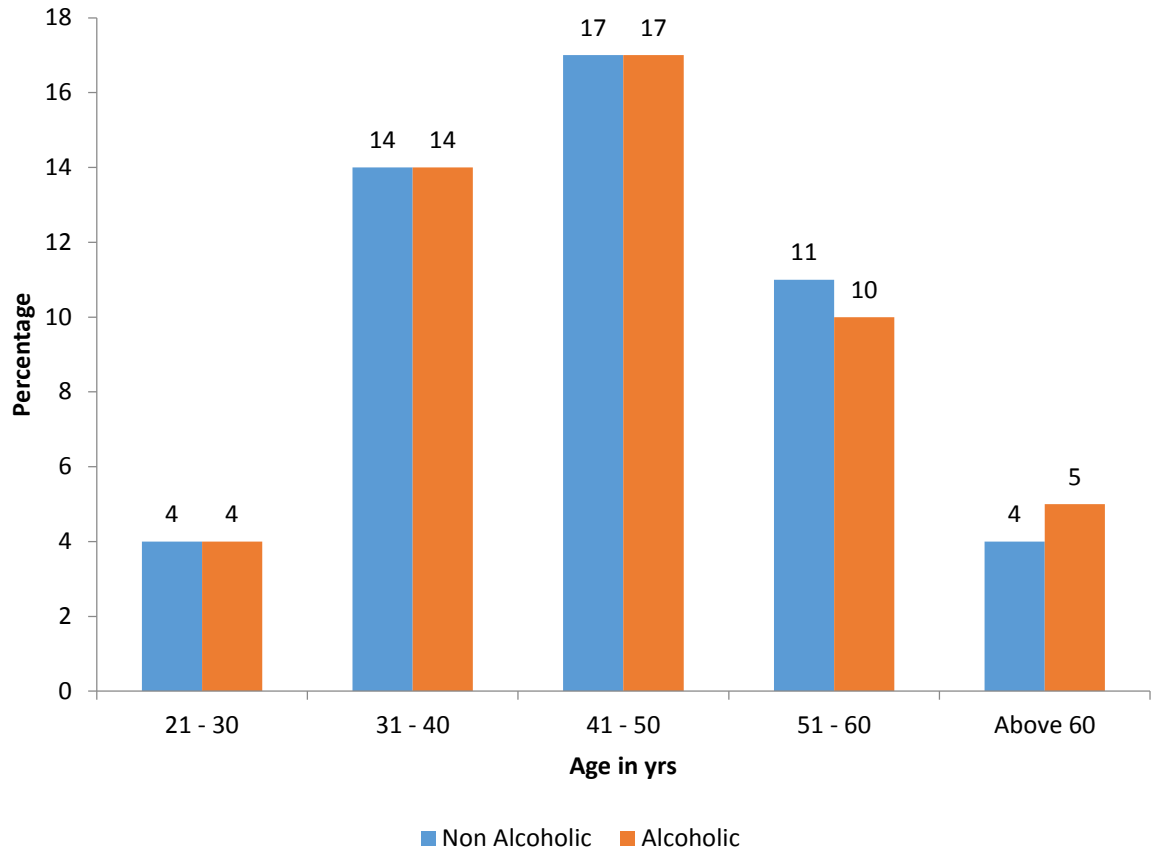
The study was conducted between August 2014 to august 2015.In our study,50 alcoholic and 50 Nonalcoholic patients admitted in the Department of General Medicine, GMKMCH, Salem were enrolled. In alcoholic group, 25 of them were moderate alcoholic and remaining 25 of them were severe alcoholic.

AGE INCIDENCE

Table – 7:

Age	Alcoholic	Non Alcoholic	Total	Chi square	p
21 - 30	4	4	8	0.16	1.00
31 - 40	14	14	28		
41 - 50	17	17	34		
51 - 60	10	11	21		
Above 60	5	4	9		
Total	**50**	**50**	**100**		

Figure: 21



Inference:

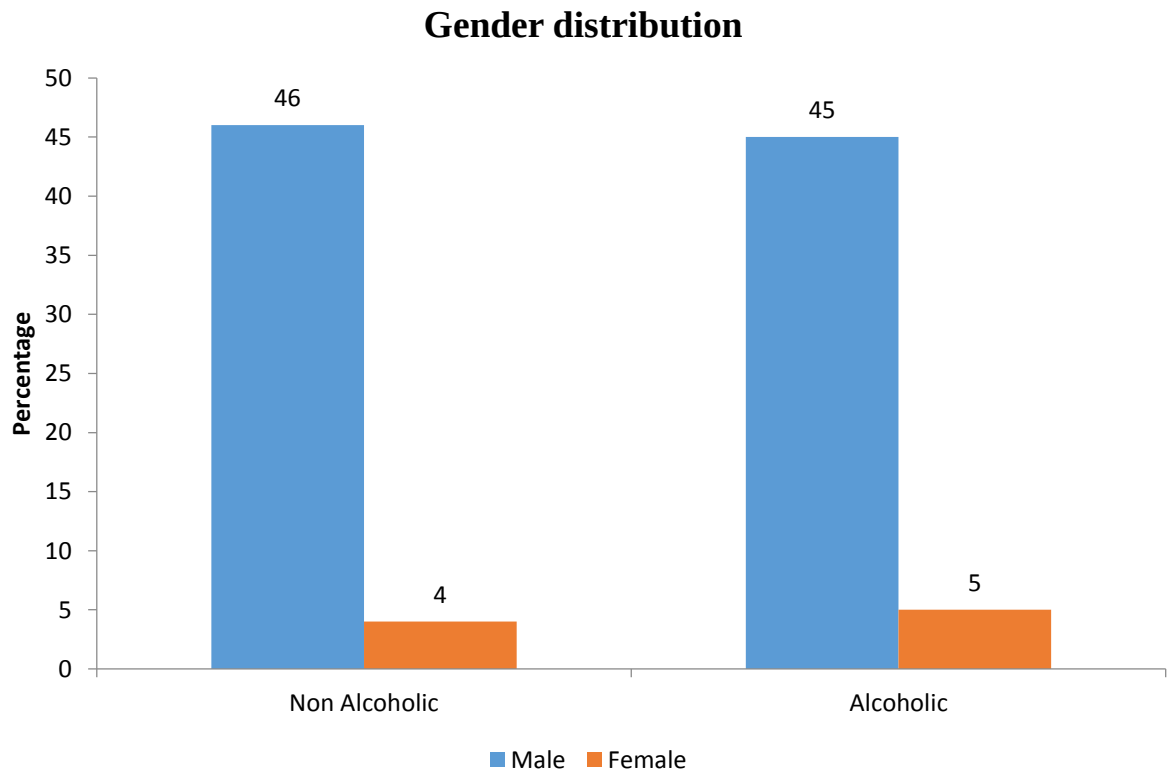
In our study, maximum number of alcoholics are in the age group (31-50), minimum number of alcoholics are in the age group (21-30). 62% of alcoholics are in the age group of 31 – 50 years. Incidence of alcohol consumption is less common below the age of 25 years and above the age of 71 years. p-value = 1 and it is statistically insignificant.

INCIDENCE OF ALCOHOLISM IN MALES Vs FEMALES

Tabel: 8

Sex	Non Alcoholic	Alcoholic	Total	Chi square	p
Male	46	45	91	0.12	0.727
Female	4	5	9		
Total	**50**	**50**	**100**		

Figure: 22



Inference:

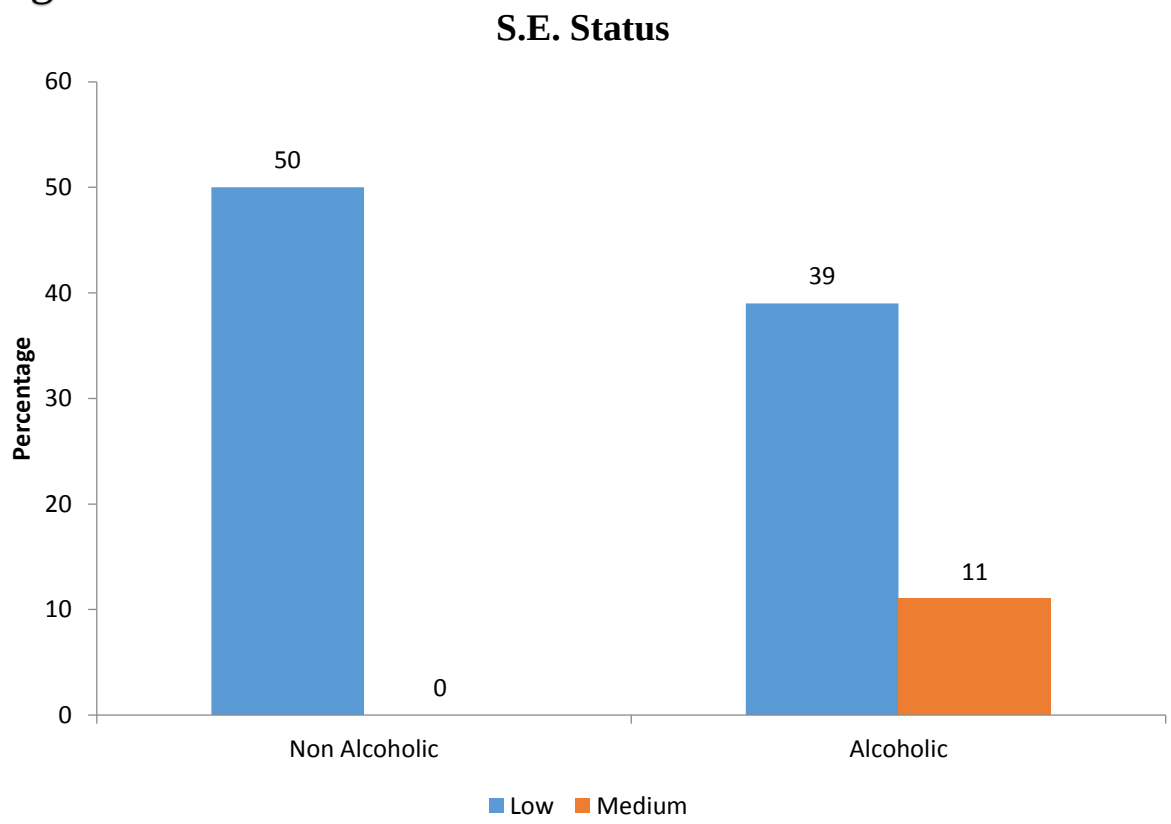
In our study, out of 50 alcoholics recruited, 45 are males (90%) and remaining 5 are female (5%). This shows that alcohol consumption is less common in females. On statistical analysis the p-value is calculated to be 0.727, is insignificant.

SOCIO-ECONOMIC STATUS

Tabel: 9

S.E. Status	Non Alcoholic	Alcoholic	Total	Chi square	p
Low	50	39	89	12.36	< 0.001**
Middle		11	11		
Total	**50**	**50**	**100**		

Figure: 23



Inference:

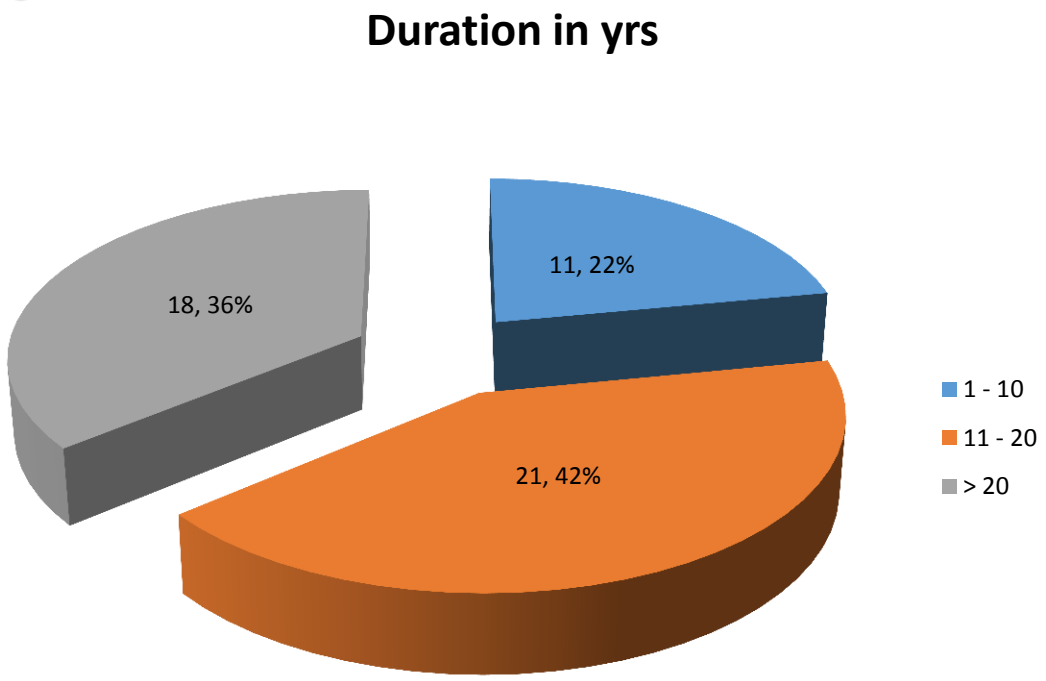
In our study, higher number of alcoholics are in low socioeconomic status, 78% remaining 22% are in middle socioeconomic status. p-value in this statistical analysis is <0.001, which is significant.

DURATION OF ALCOHOL INTAKE

Table: 10

Duration	Frequency	Percent
1 - 10	11	22
11- 20	21	42
> 20	18	36
Total	**50**	**100**

Figure: 24



Inference:

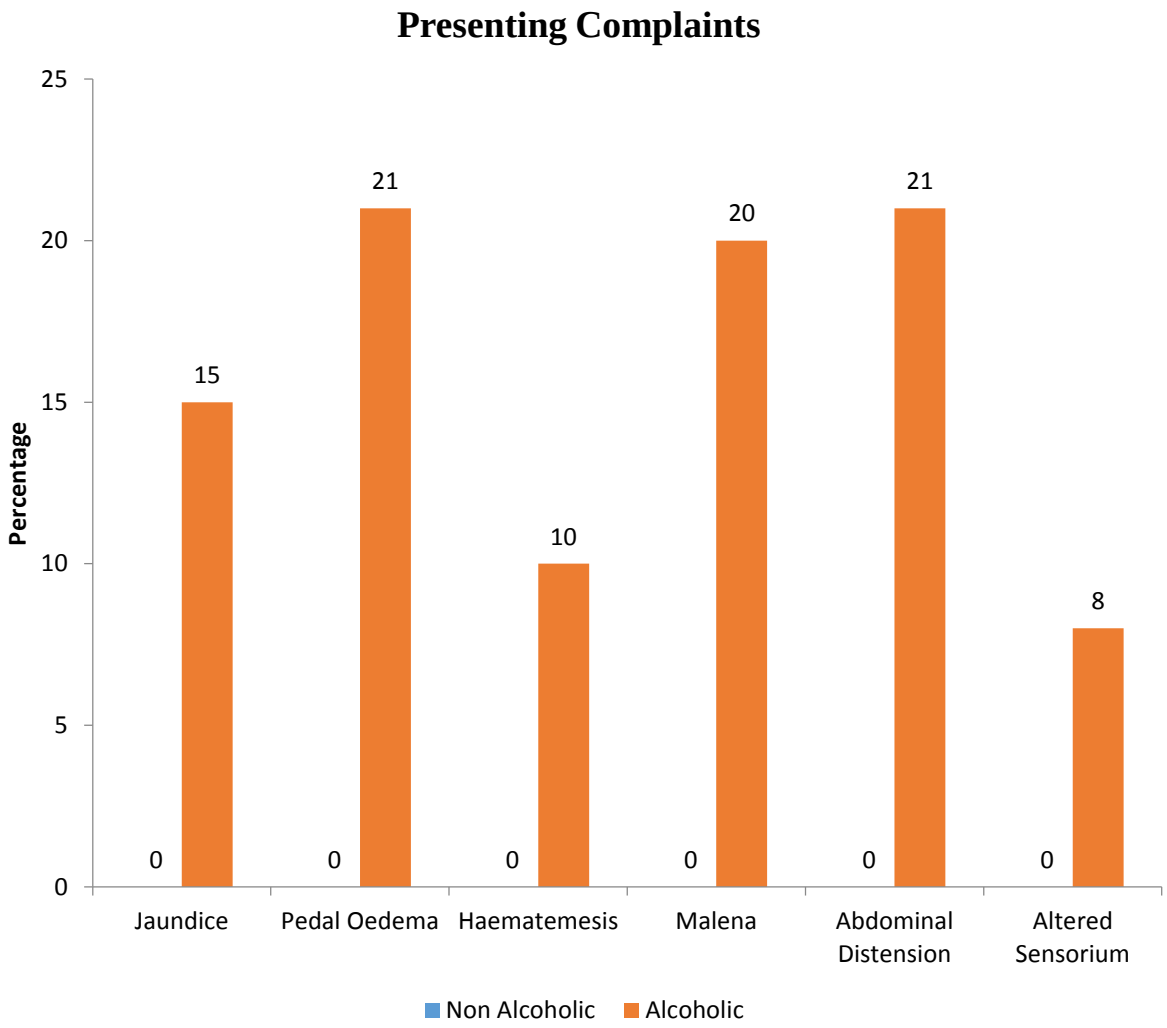
In present study, 42% of patients in study group are alcoholic for a period of 11-20 years, 36% are alcoholic for a period of more than 20 years and the remaining 22% of them were alcoholic for 10 years and below.

SYMPTOM ANALYSIS OF PATIENTS

Table: 11

Presenting Complaints		Alcoholic		Total	Chi square	p
		Non Alcoholic	Alcoholic			
Jaundice	No	50	35	85	17.65	< 0.001**
	Yes		15	15		
Pedal Oedema	No	50	29	79	26.58	< 0.001**
	Yes		21	21		
Haematemesis	No	50	40	90	11.11	0.001**
	Yes		10	10		
Malena	No	50	30	80	25.00	< 0.001**
	Yes		20	20		
Abdominal Distension	No	50	29	79	26.58	< 0.001**
	Yes		21	21		
Altered Sensorium	No	50	42	92	8.70	0.003**
	Yes		8	8		
Total		50	50	100		

Figure: 25



Inference:

Among 50 alcoholics, abdominal distension is present in 42% of patients, pedal oedema in 42% of patients, malena in 40% of patients, Jaundice in 30% of patients, Haematemesis in 20% of patients and altered sensorium in 16% of patients. In our study, most common presentation of

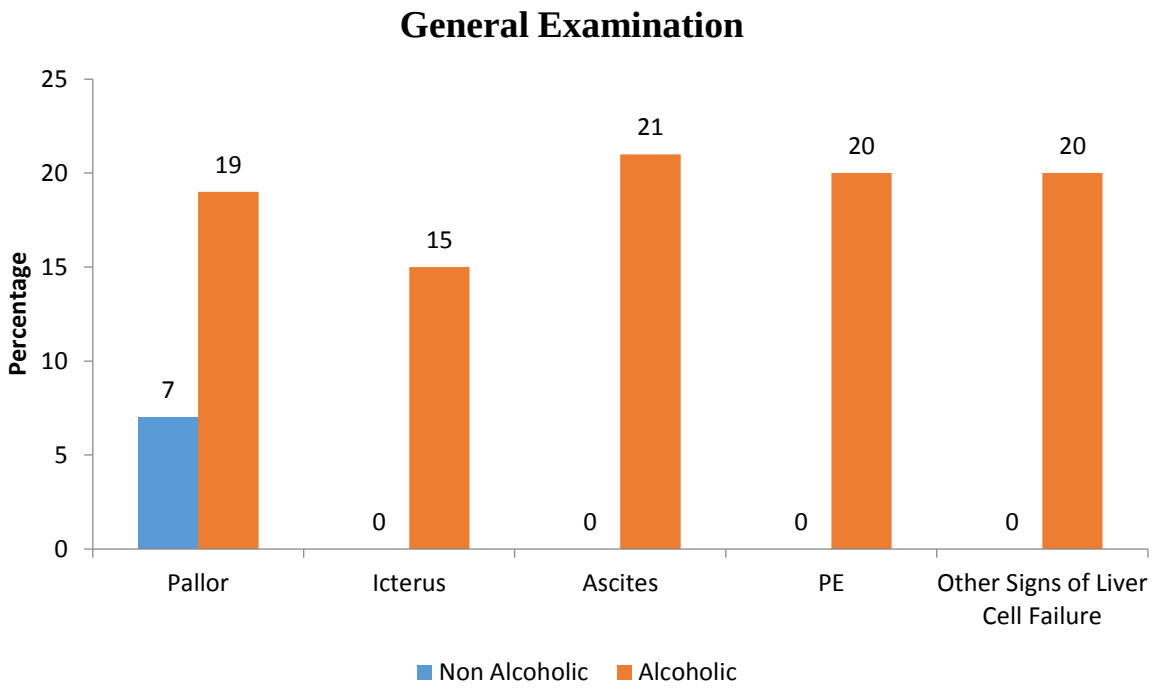
alcoholics is abdominal distension and pedal oedema, followed by malena. Jaundice and Haematemesis are less common. (p-value <0.001)

GENERAL EXAMINATION COMPARISON

Table: 12

Presenting Complaints		Alcoholic		Total	Chi square	p
		Non Alcoholic	Alcoholic			
Pallor	No	43	31	74	7.48	0.006**
	Yes	7	19	26		
Icterus	No	50	35	85	17.65	< 0.001**
	Yes		15	15		
Ascites	No	50	29	79	26.58	< 0.001**
	Yes		21	21		
PE	No	50	30	80	25.00	< 0.001**
	Yes		20	20		
Other Signs of Liver Cell Failure	No	50	30	80	25.00	< 0.001**
	Yes		20	20		
Total		50	50	100		

Figure: 26



Inferenece:

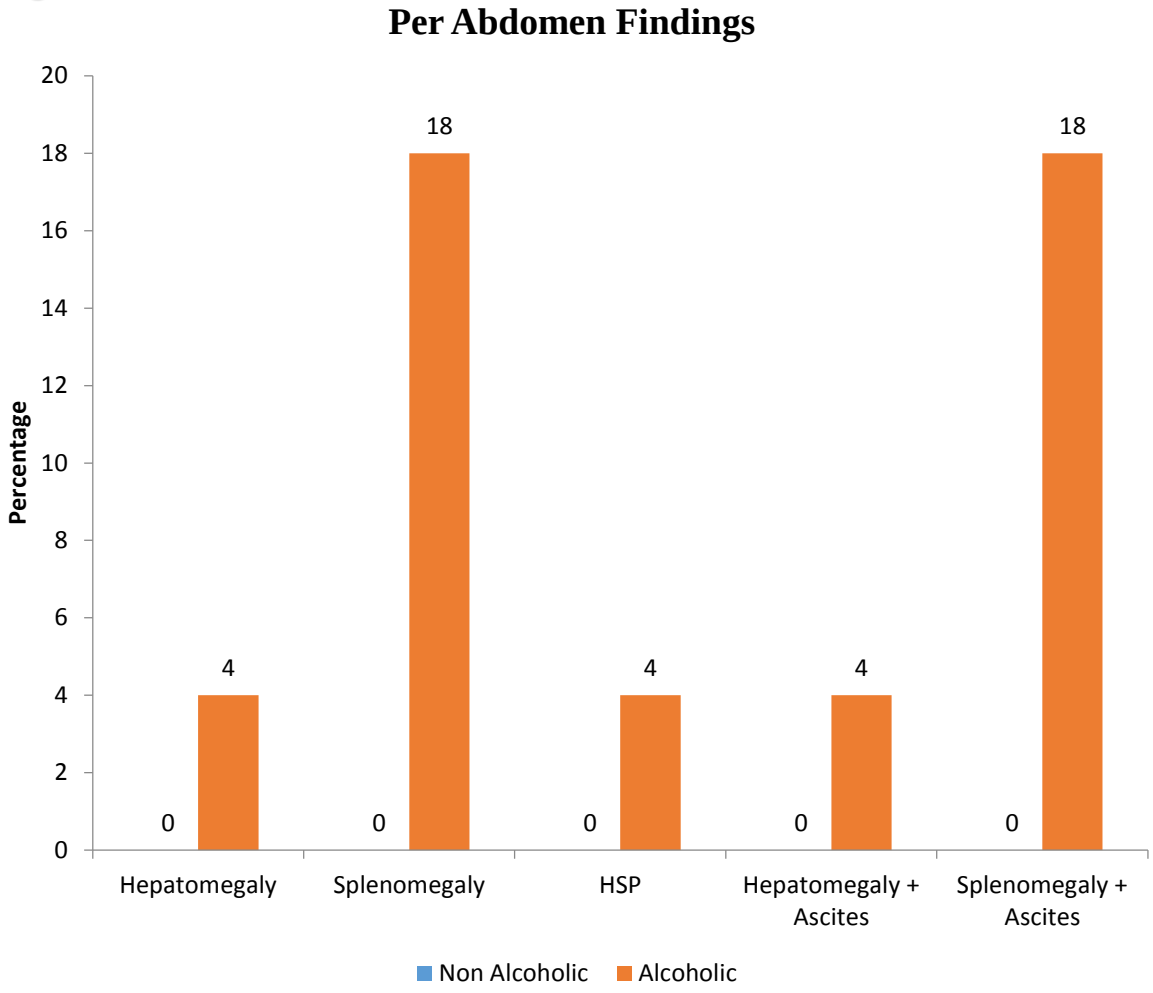
In our study pallor is present in 38% of alcoholics and 14% of non-alcoholics. This shows that Anemia is common in alcoholics, when compared with non-alcoholics icterus is present in 30%, Ascites in 42%, pedal oedema in 40%, other signs of Hepatic failure such as loss of axillary hair, testicular atrophy are seen in 40% of alcoholics.

PER ABDOMEN EXAMINATION

Table: 13

Per Abdomen Findings		**Non Alcoholic**	**Alcoholic**	**Total**	**Chi square**	**p**
Hepatomegaly	No	50	46	96	4.17	0.041*
	Yes		4	4		
Splenomegaly	No	50	32	82	21.95	< 0.001**
	Yes		18	18		
HSP	No	50	46	96	4.17	0.041*
	Yes		4	4		
Hepatomegaly + Ascites	No	50	46	96	4.17	0.041*
	Yes		4	4		
Splenomegaly + Ascites	No	50	32	82	21.95	< 0.001**
	Yes		18	18		
Total		**50**	**50**	**100**		

Figure: 27



Inference:

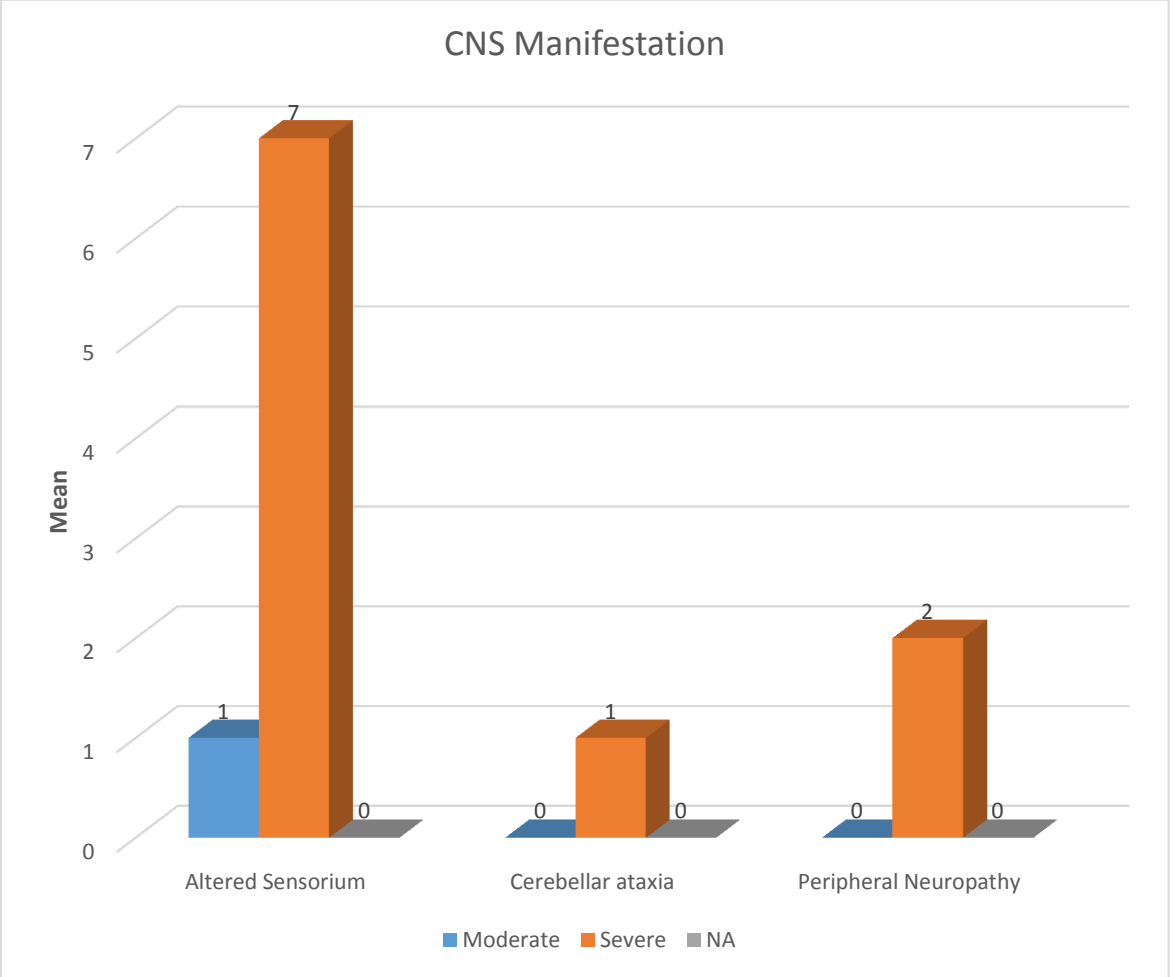
In our study, Hepatomegaly alone is present in 8% of alcoholics. Splenomegaly is present in 36%, both Hepatomegaly and Splenomegaly is 8%, Hepatomegaly and Ascites is 8% and Splenomegaly and Ascites in 36% of alcoholics.

CNS MANIFESTATION

Table: 14

CNS Manifestation	Alcoholic			Chi Square	p value
	Moderate	Severe	NA		
Altered Sensorium	1	7	0	8.70	0.003
Cerebellar ataxia	0	1	0	3.03	0.220
Peripheral Neuropathy	0	2	0	4.12	0.159

Figure: 28



Inference:

In the present study, 16% presented with altered sensorium, Cerebellar ataxia in 2% of alcoholics and Peripheral neuropathy in 4% of alcoholics.

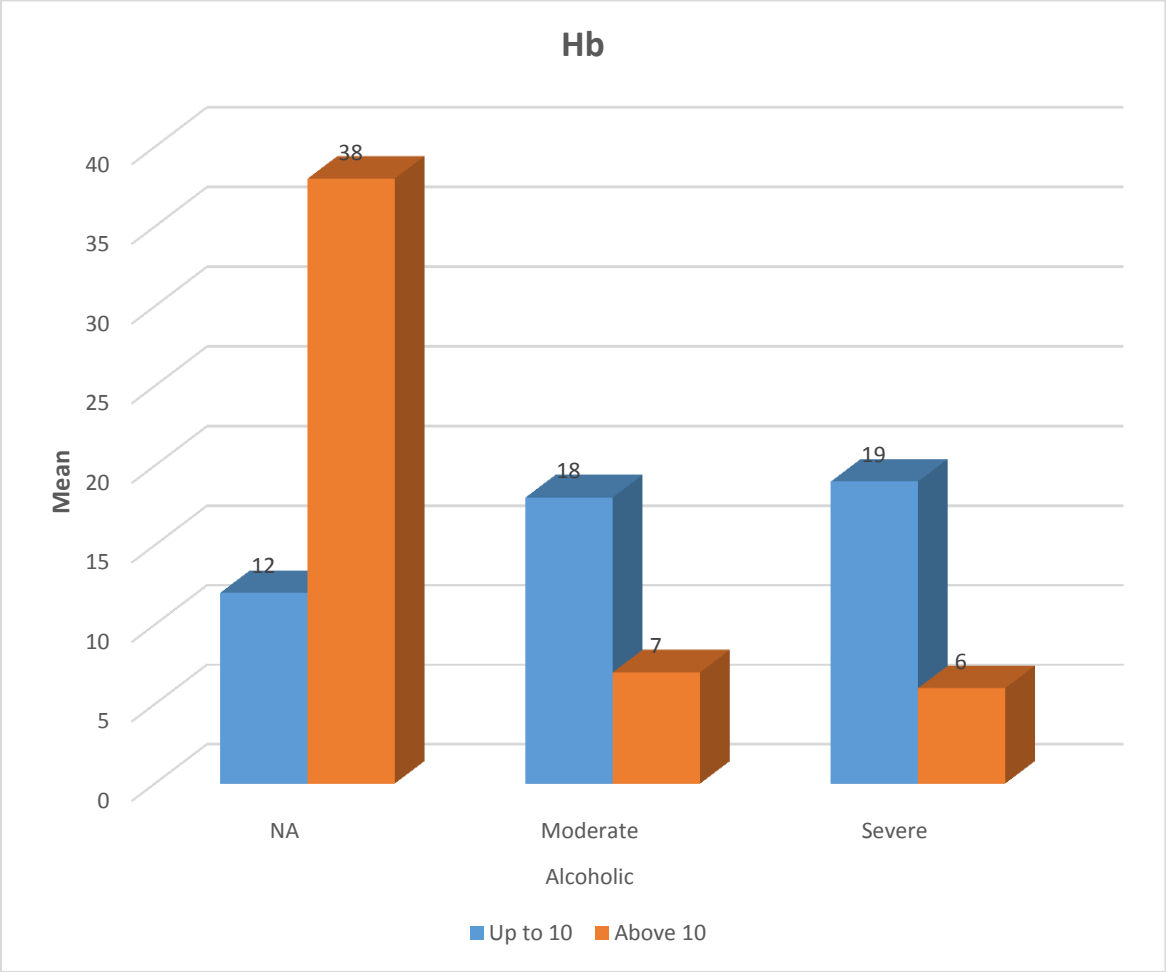
LEVEL OF HEMOGLOBIN IN MODERATE AND SEVERE ALCOHOLICS

Table: 15

Hb	Frequency	Percent
Up to 10	49	49
Above 10	51	51
Total	100	100

Hb	Alcoholic			Total	Chi square	p
	NA	Moderate	Severe			
Up to 10	12	18	19	49	25.09	
Above 10	38	7	6	51		
Total	**50**	**25**	**25**	**100**		

Figure: 29



Inference:

In our study, the Hemoglobin Level up to 10gms% present in 76% of severe alcoholic. Only 24% of severe alcoholic have Hb lever of more than 10gms%. 72% of moderate alcoholics have Hb 10gm and below, 28% of moderate alcoholic have Hb more than 10gms. 76% of non-alcoholics have Hb level above 10gms%. Only 24% of non-alcoholic

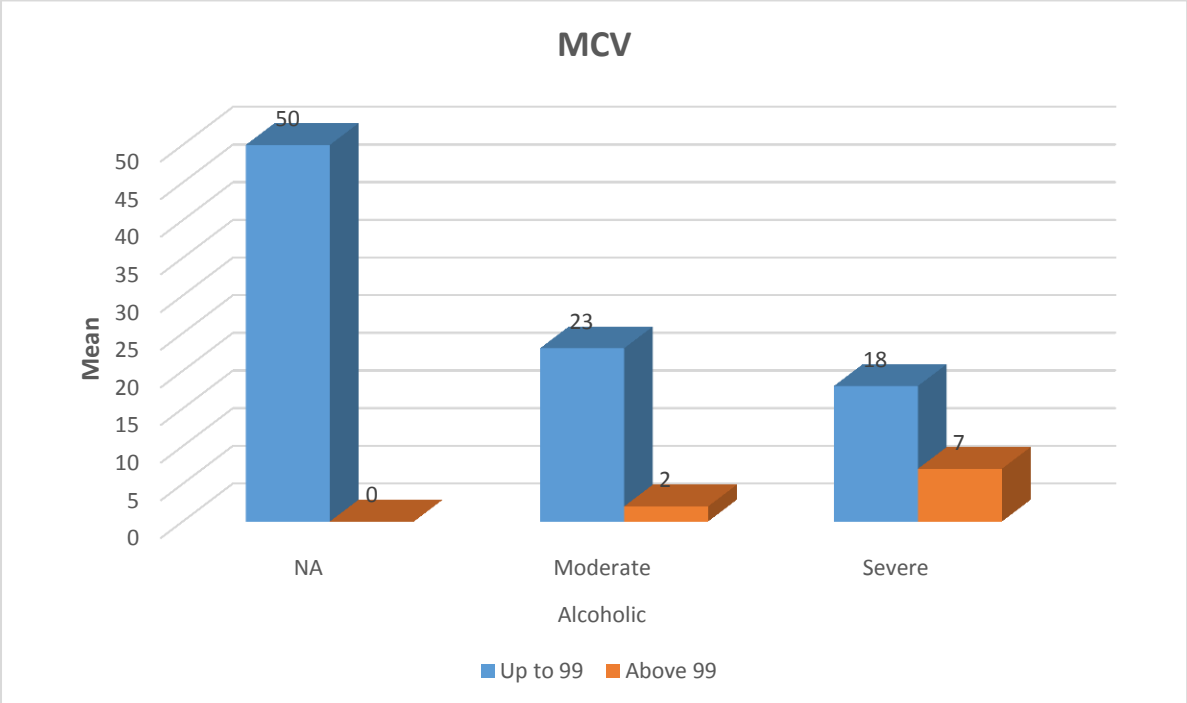
have Hb 10gm and below. 74% of alcoholics are anemic. Even 24% of non-alcoholics are anemic. Minimum Hb value is 2 gms% and highest is 16gms%.

MCV

Table: 16

MCV	Alcoholic			Total	Chi square	P value
	NA	Moderate	Severe			
Up to 99	50	23	18	91	16.00	
Above 99	0	2	7	9		
Total	**50**	**25**	**25**	**100**		

Figure: 30



Inference:

In the present study, MCV >99 fl is present in 28%, 8% and of 0% of severe, moderate alcoholics and non-alcoholic respectively. MCV of up to 99fl is present in 72%, 92% and 100% of severe, Moderate and non-alcoholic respectively.18% of alcoholics has MCV more than 99fl. Lowest MCV value is 75fl and highest value is 116fl.

Table: 17

		N	Mean	SD	ANOVA	p
Hb	NA	50	11.12	2.24	9.30	< 0.001**
	Moderate	25	9.33	1.20		
	Severe	25	9.37	2.30		
	Total	100	10.24	2.21		
RBC	NA	50	3.90	0.58	15.04	< 0.001**
	Moderate	25	3.26	0.74		
	Severe	25	3.13	0.68		
	Total	100	3.55	0.74		
PCV	NA	50	38.72	2.78	52.50	< 0.001**
	Moderate	25	33.55	4.26		
	Severe	25	28.30	6.14		
	Total	100	34.82	6.02		
MCV	NA	50	89.56	3.84	6.24	0.003
	Moderate	25	85.60	8.35		
	Severe	25	93.44	12.21		
	Total	100	89.54	8.25		
MCH	NA	50	30.54	2.27	37.23	< 0.001**
	Moderate	25	25.48	1.62		
	Severe	25	28.91	3.18		
	Total	100	28.87	3.15		
MCHC	NA	49	36.18	1.92	35.74	< 0.001**
	Moderate	25	32.57	1.94		
	Severe	25	33.24	2.00		
	Total	99	34.52	2.54		
T.C	NA	50	8296.00	1762.30	8.83	< 0.001**
	Moderate	25	7872.00	3537.72		
	Severe	25	11072.00	4286.17		

	Total	100	8884.00	3266.04		
Platelet	NA	50	2.50	0.37	39.93	< 0.001**
	Moderate	25	1.63	0.66		
	Severe	25	1.47	0.67		
	Total	100	2.03	0.72		

Figure: 31

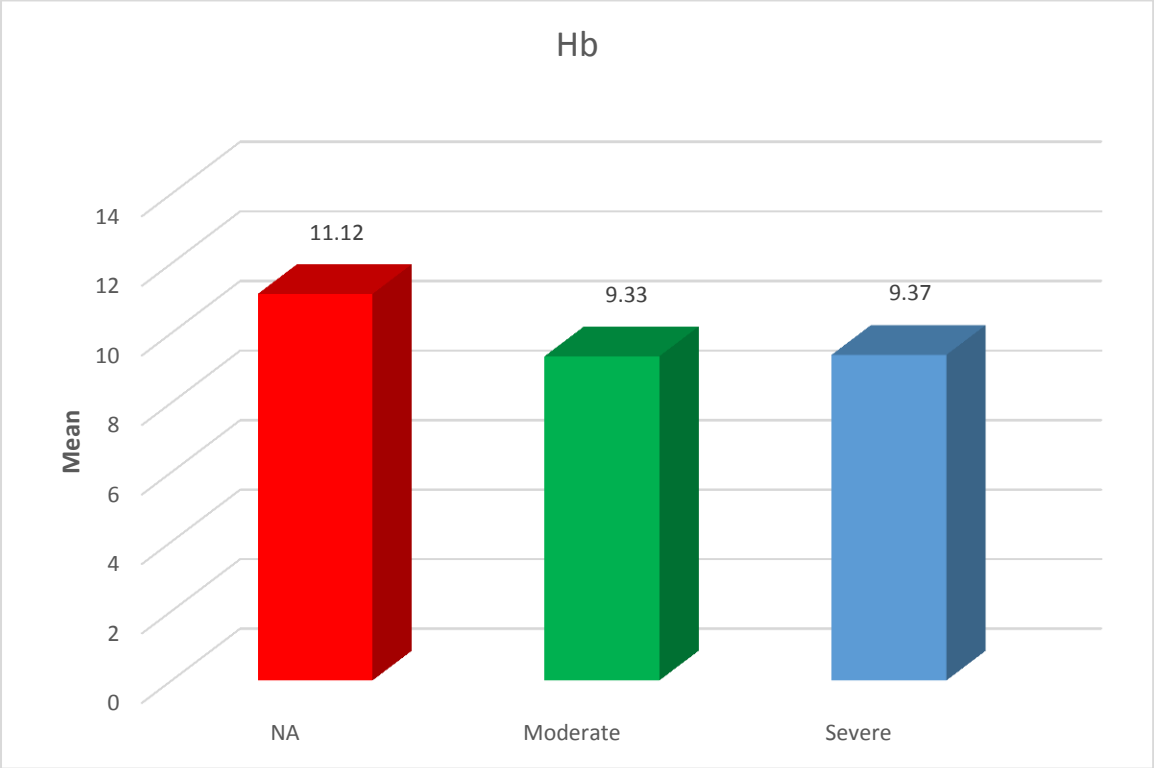


Figure: 32

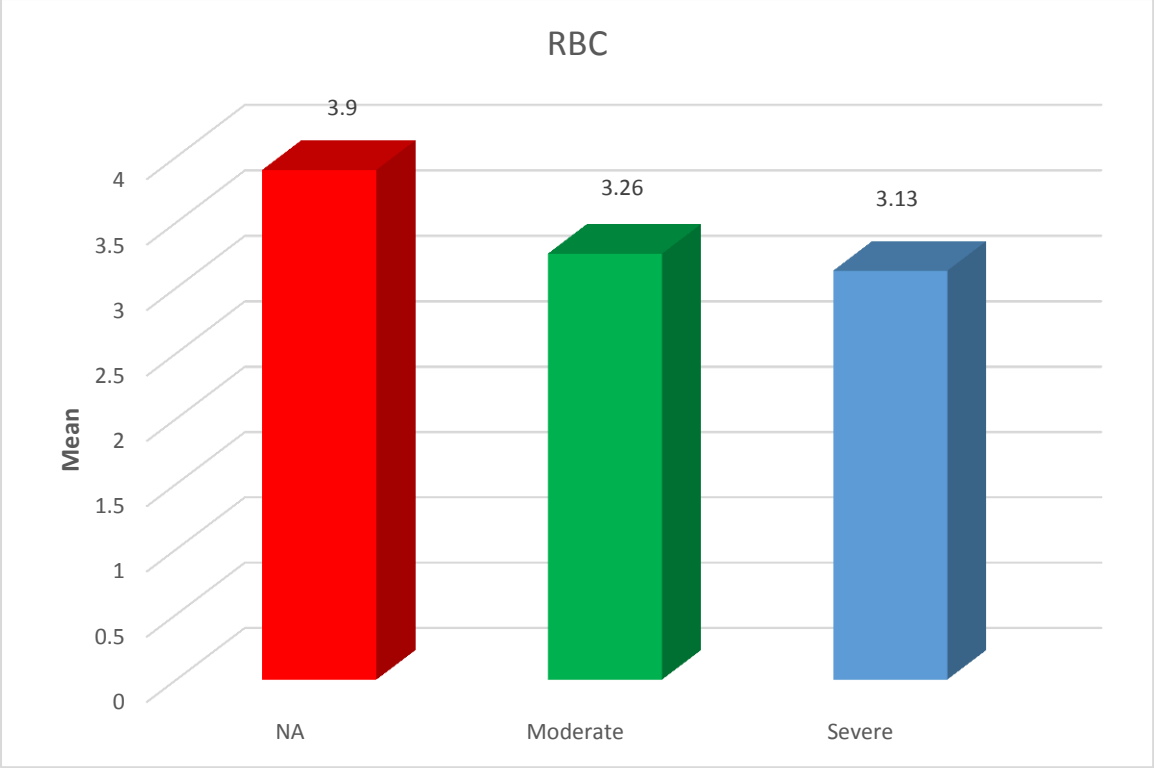


Figure: 33

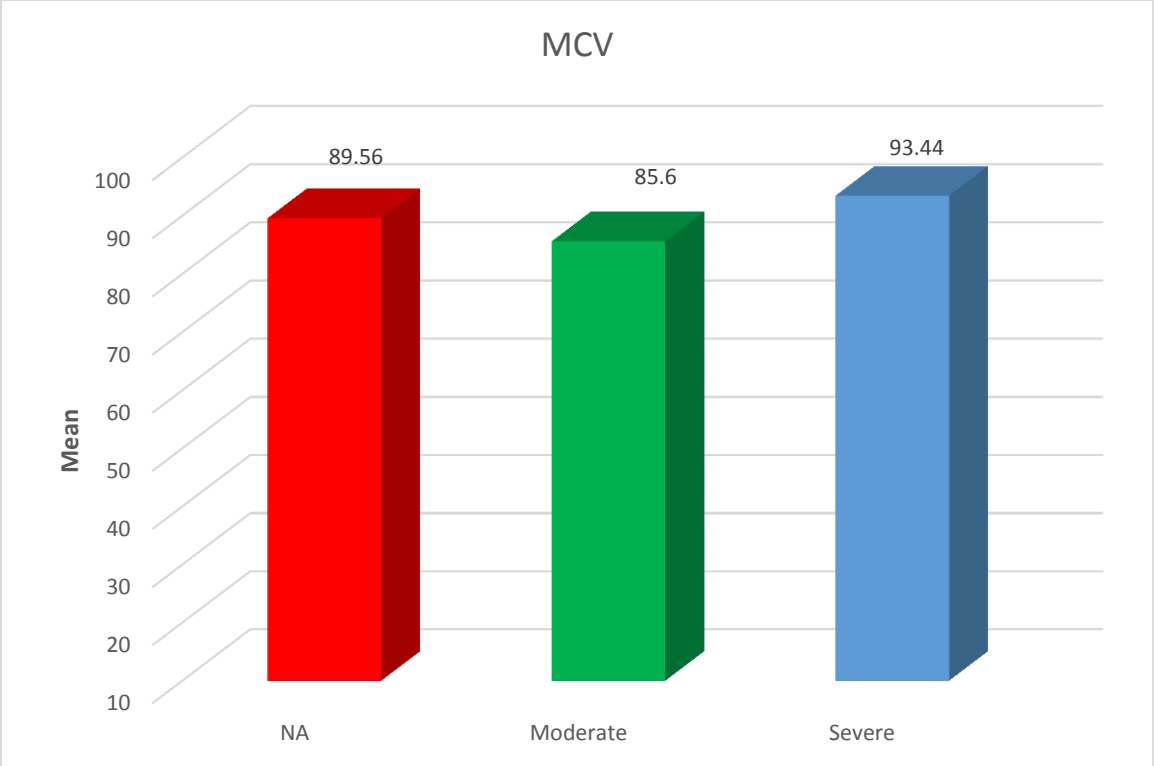


Figure: 34

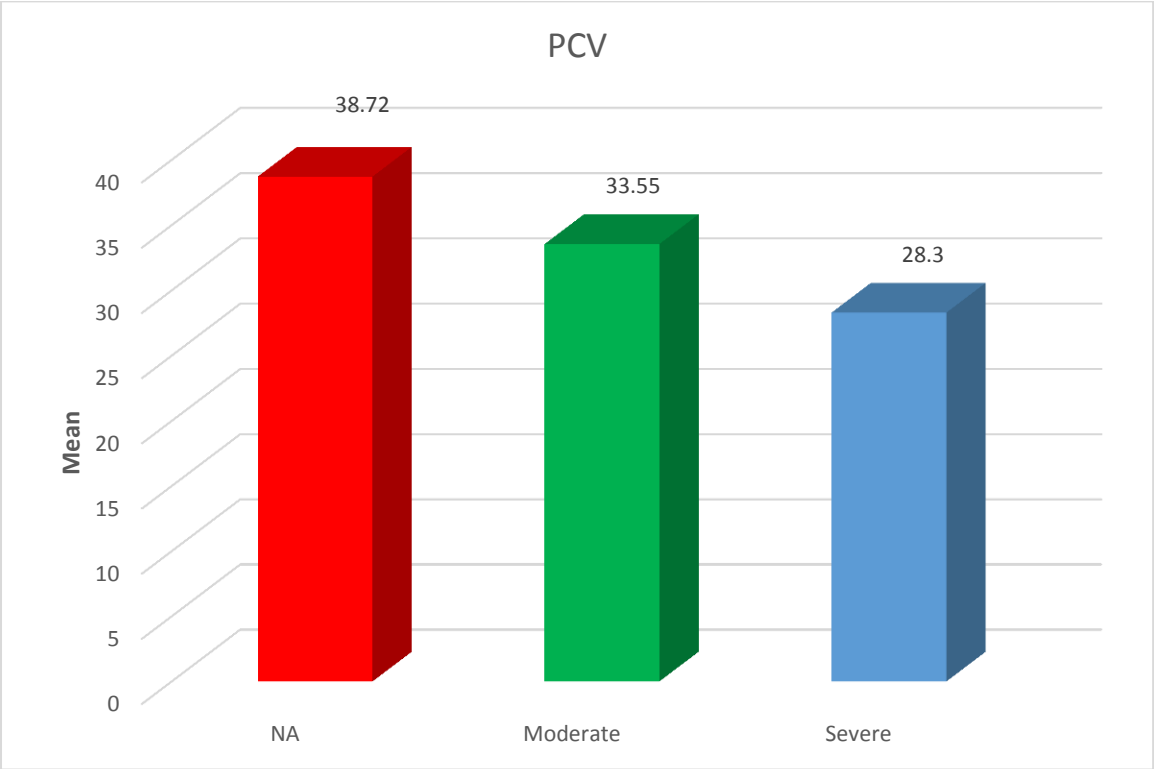


Figure: 35

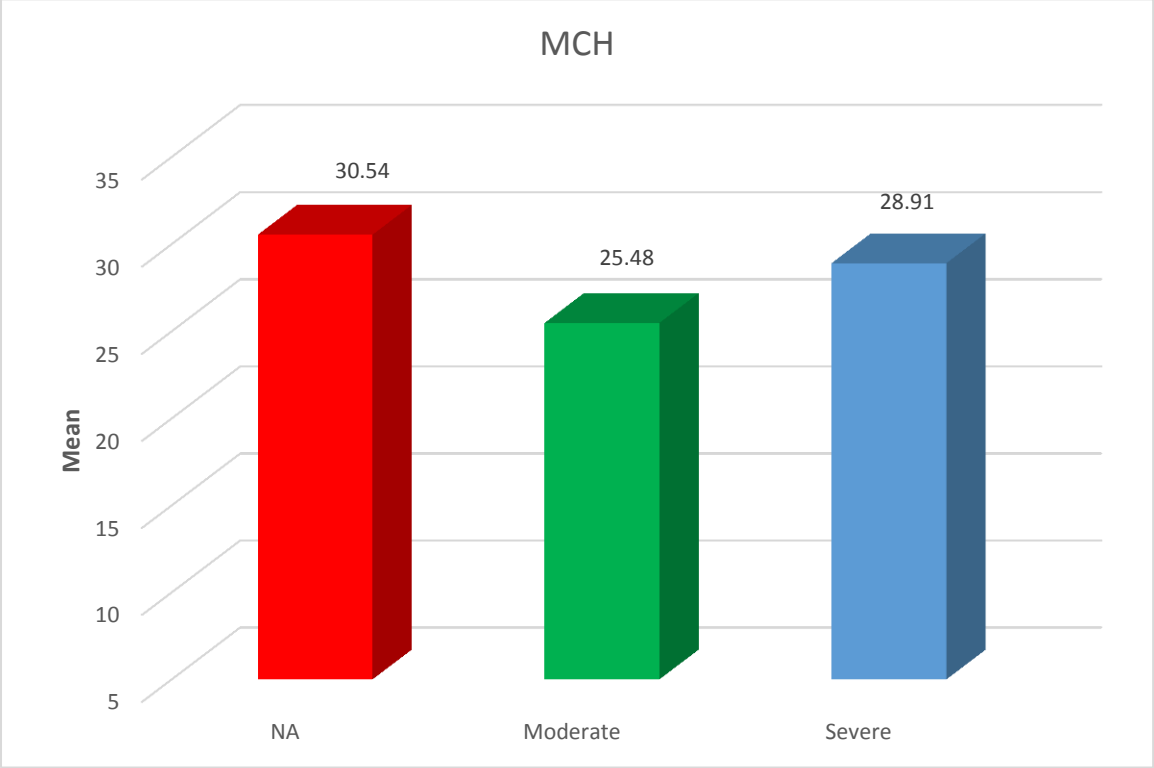


Figure: 36

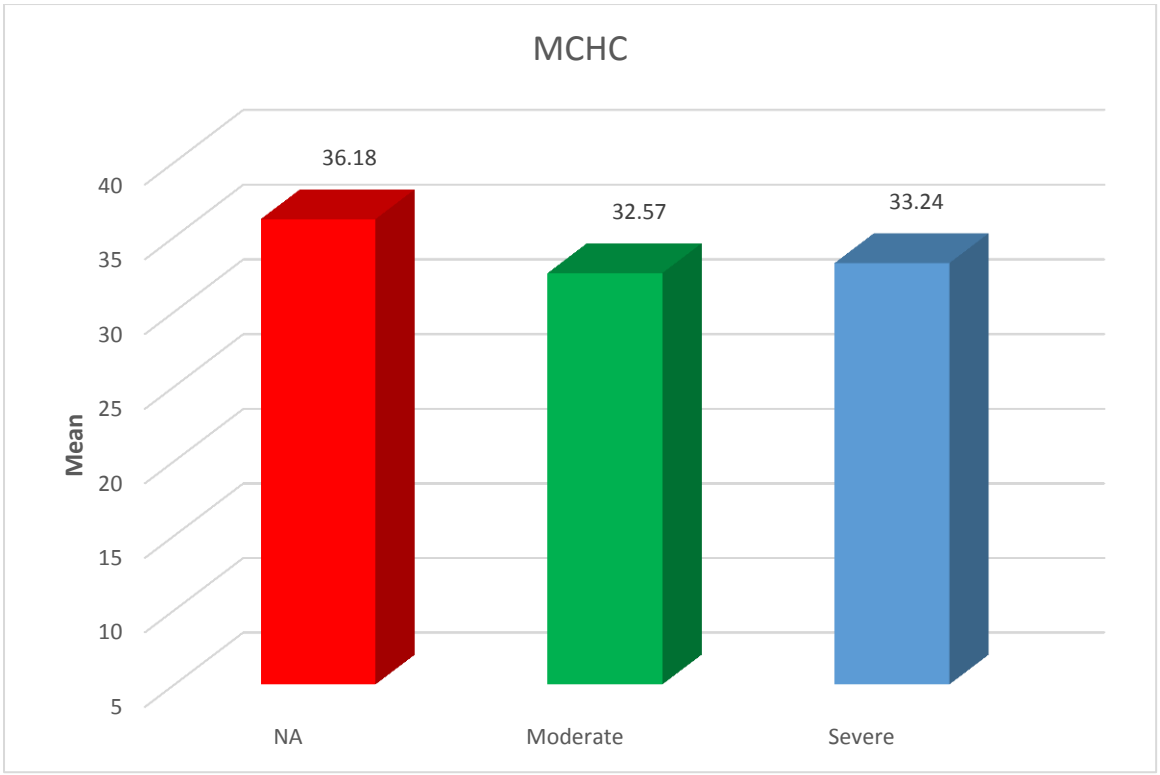
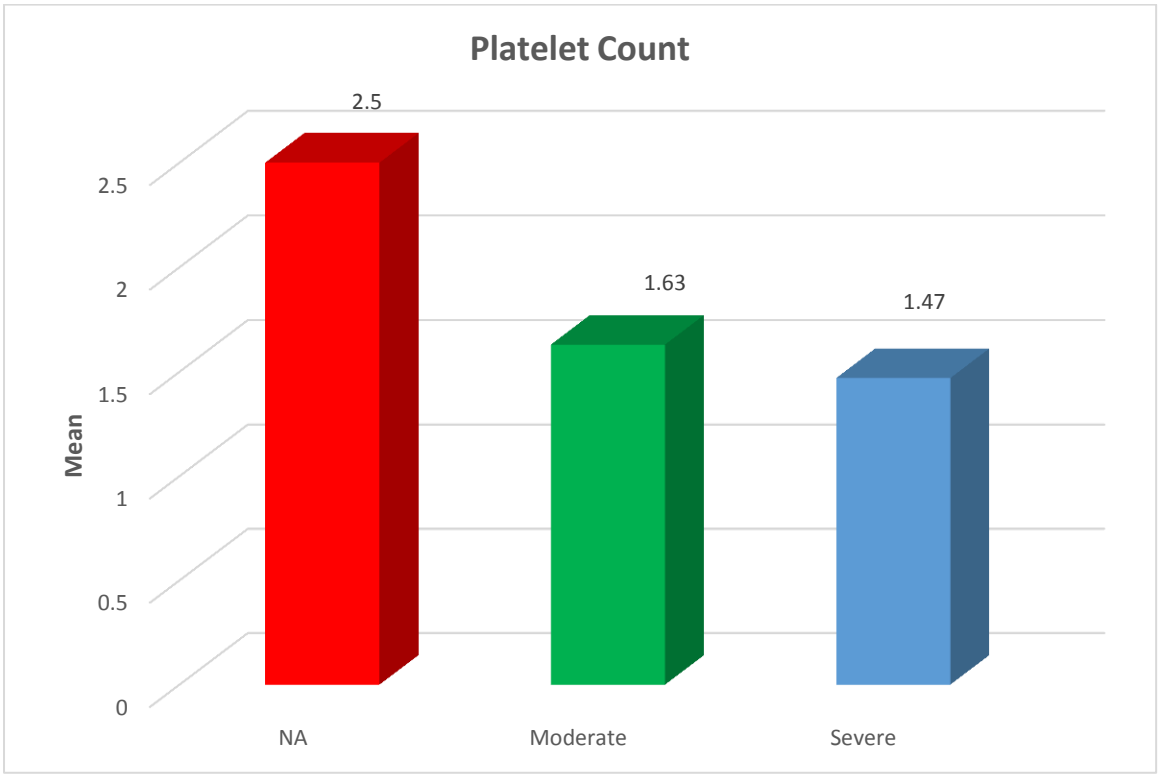

MCHC
36.18
32.57
33.24
40
35
30
25
20
15
10
5
Mean
NA
Moderate
Severe

Figure: 37

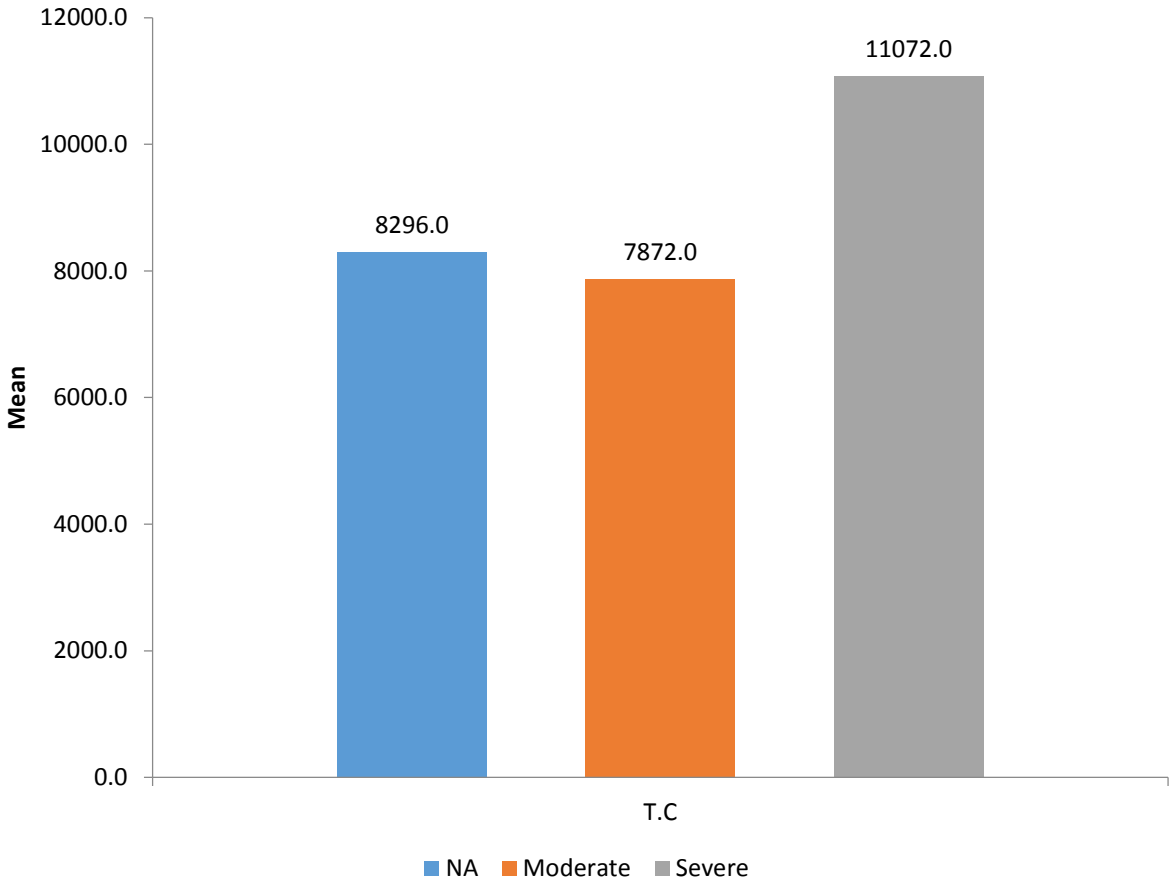

Platelet Count
2.5
1.63
1.47
2.5
2
1.5
1
0.5
0
Mean
NA
Moderate
Severe

Mean Hb level in moderate drinker is 9.33 ± 2.30. (p-value <0.001) RBC count mean is 3.26 ± 0.74, 3.13 ± 0.68 in moderate and severe drinkers respectively. Mean MCV is 85.60 ± 8.37, 93.44 ± 12.21 in moderate and severe drinkers respectively MCH is 25.48 ± 1.62 and 28.91 ± 3.18 MCHC is 32.57 ± 1.94, 33.24 ± 2.00 in moderate and severe drinkers respectively.

Platelet count is 1.63 ± 0.66, 1.47 ± 0.67 in moderate and severe drinkers in our study. Lowest MCV in our study is 75 fl and Maximum value is 116fl. Total count lowest value is 2900 cells and maximum count is 26200 cells in our study.

Figure: 38

WBC's COUNT



Inference:

Mean WBC Count of severe alcoholics is 11072 ± 4286.17, moderate alcoholics is 7872.00 ± 3537.72, non-alcoholic is 8296 ± 1762.30 Leukocytosis in severe alcoholics is due to infection. (p-value is <0.001).

Table: 18 Liver Function Test

		N	Mean	SD	ANOVA	p
T. Bilirubin	NA	50	0.97	0.20	23.50	< 0.001**
	Moderate	25	2.52	2.38		
	Severe	25	3.95	2.73		
	Total	100	2.10	2.18		
D. Bilirubin	NA	50	0.32	0.08	23.95	< 0.001**
	Moderate	25	0.86	0.77		
	Severe	25	1.28	0.87		
	Total	100	0.70	0.71		
Indirect Bilirubin	NA	50	0.65	0.16	22.93	< 0.001**
	Moderate	25	1.65	1.61		
	Severe	25	2.67	1.88		
	Total	100	1.41	1.48		
T. Protein	NA	50	7.44	0.46	30.60	< 0.001**
	Moderate	24	6.68	0.39		
	Severe	25	6.92	0.37		
	Total	99	7.13	0.54		
Albumin	NA	50	4.44	0.51	67.44	< 0.001**
	Moderate	25	3.55	0.31		
	Severe	25	3.40	0.28		
	Total	100	3.96	0.64		
SGOT	NA	50	20.16	4.11	61.38	< 0.001**
	Moderate	25	67.40	27.45		
	Severe	25	94.44	50.51		
	Total	100	50.54	42.83		
SGPT	NA	50	27.40	5.08	47.76	< 0.001**
	Moderate	25	48.84	15.33		
	Severe	25	57.44	21.27		
	Total	100	40.27	18.87		
Alk. Phosphatas	NA	50	51.92	10.47	2.53	0.085
	Moderate	25	48.16	8.28		
	Severe	25	54.52	10.89		
	Total	100	51.63	10.24		

Figure: 39

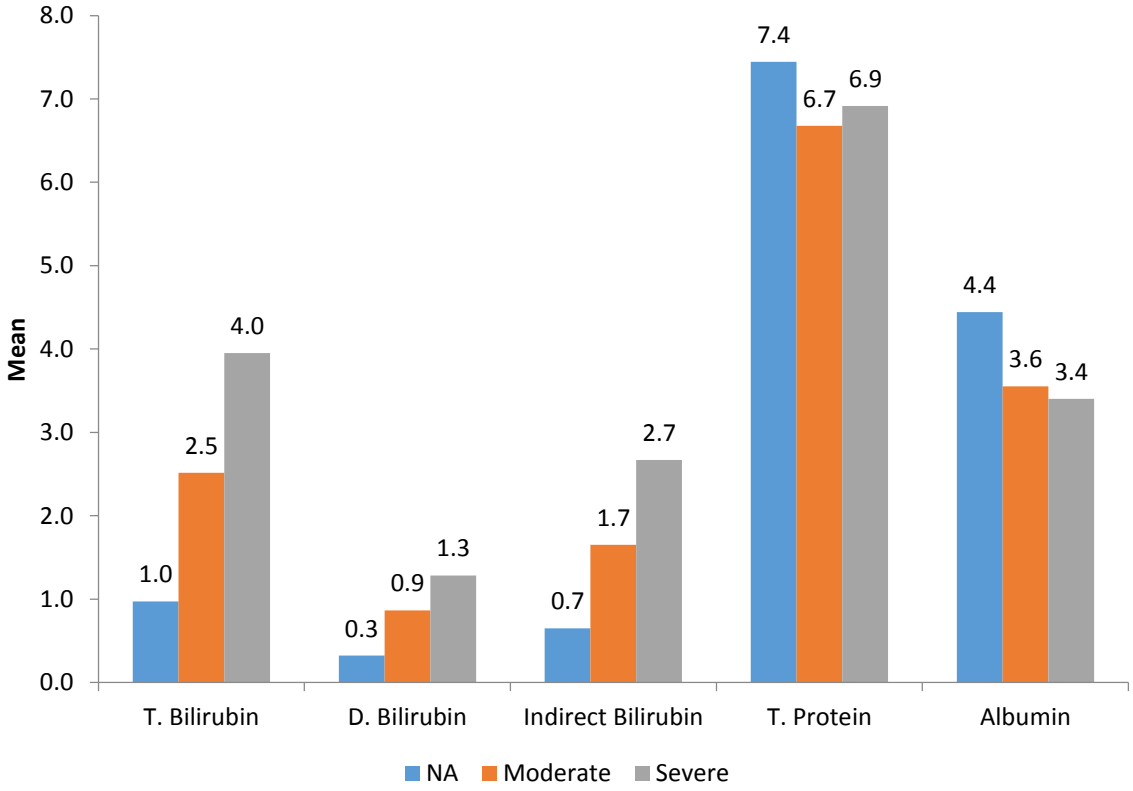
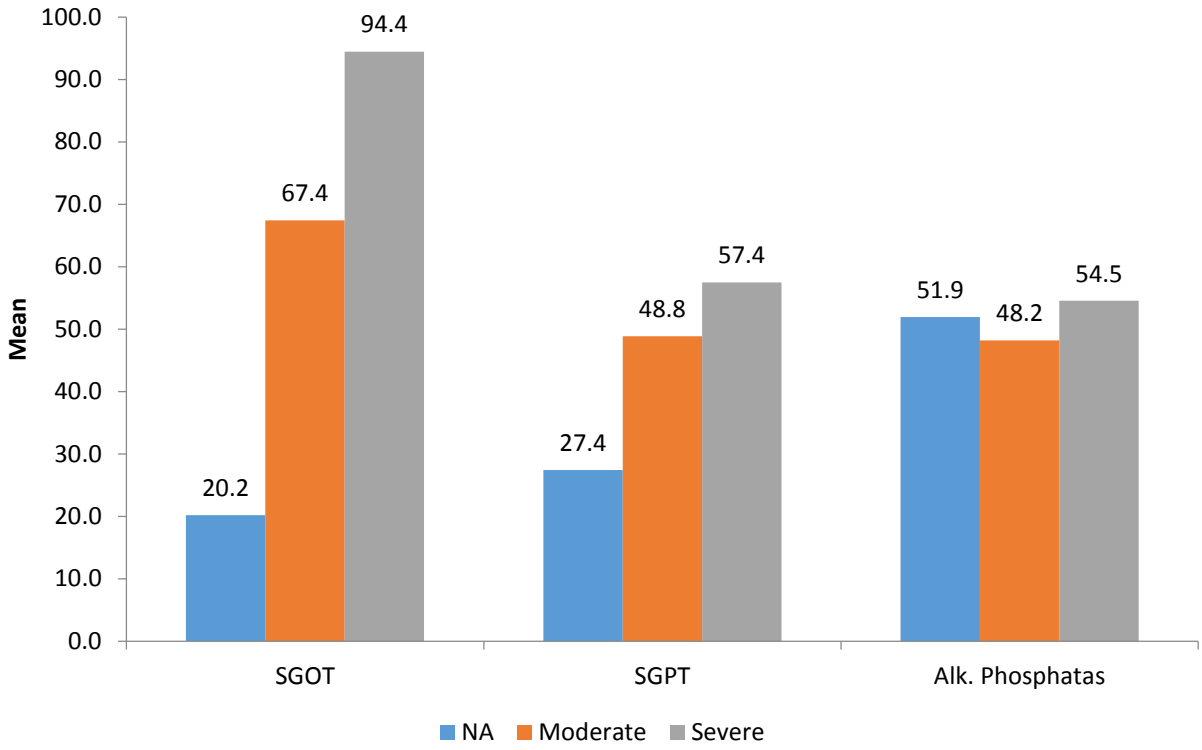

8.0
7.0
6.0
5.0
4.0
3.0
2.0
1.0
0.0
Mean
1.0
2.5
4.0
0.3
0.9
1.3
0.7
1.7
2.7
7.4
6.7
6.9
4.4
3.6
3.4
T. Bilirubin
D. Bilirubin
Indirect Bilirubin
T. Protein
Albumin
NA
Moderate
Severe

Figure: 40



Inference:

In the present study, the mean total Bilirubin is 2.52 ± 2.38, 3.95 ± 2.73 in moderate and severe drinkers respectively. Mean serum albumin is 3.55 ±0.31 and 3.40 ± 0.28 in moderate and severe alcoholics respectively.

Mean SGOT level is 94.44 ± 50.51 in severe alcoholics and SGPT mean value is 57.44 ± 21.27 in them. SGOT mean value in moderate drinkers is 67.40 ± 27.45 and SGPT is 48.84 ± 15.33. Elevated Alkaline phosphate in alcoholics is statistically not significant.

BLOOD UREA AND SERUM CREATININE

Table: 19

		N	Mean	SD	ANOVA	p
Blood Urea	NA	50	27.00	6.49	150.98	< 0.001**
	Moderate	25	79.68	17.88		
	Severe	25	95.68	29.49		
	Total	100	57.34	35.65		
Serum Creatinine	NA	50	0.94	0.77	28.16	< 0.001**
	Moderate	25	2.04	0.88		
	Severe	25	3.06	1.90		
	Total	100	1.75	1.47		

Figure: 41

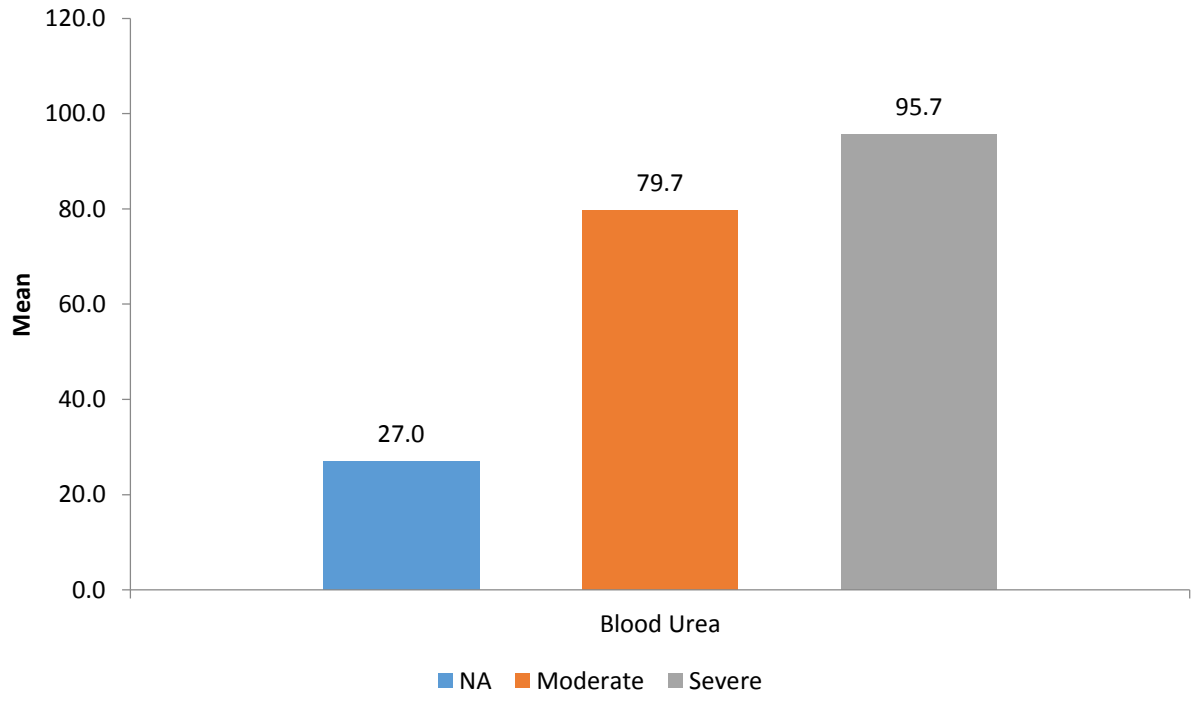
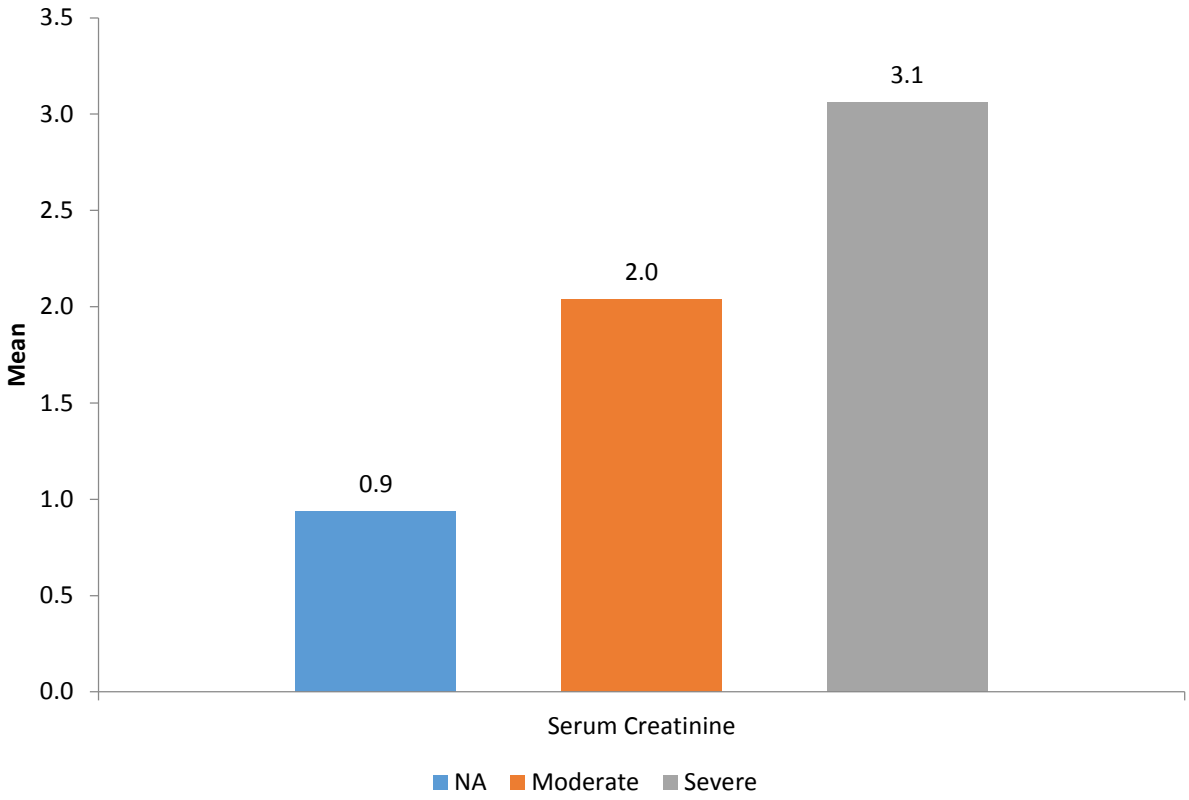


Figure: 42



Inference:

In present study, mean blood urea level is 79.68 ± 17.88 in moderate drinkers, 95.68 ± 29.49 in severe drinkers. Serum Creatinine mean value is 2.04 ± 0.88 and 3.06 ± 1.90 in moderate and severe alcoholics respectively with p-value of <0.001.

Table: 20

		N	Mean	SD	ANOVA	p
P.T	NA	50	14.08	0.83	149.16	< 0.001**
	Moderate	25	25.76	6.41		
	Severe	20	31.35	5.39		
	Total	95	20.79	8.42		
Vit. B12	NA	50	605.12	132.53	47.77	< 0.001**
	Moderate	25	713.80	374.14		
	Severe	25	1375.60	510.03		
	Total	100	824.91	458.03		
Folic acid	NA	50	12.18	1.72	91.17	< 0.001**
	Moderate	25	8.71	1.00		
	Severe	25	8.08	1.01		
	Total	100	10.29	2.37		

Figure: 43

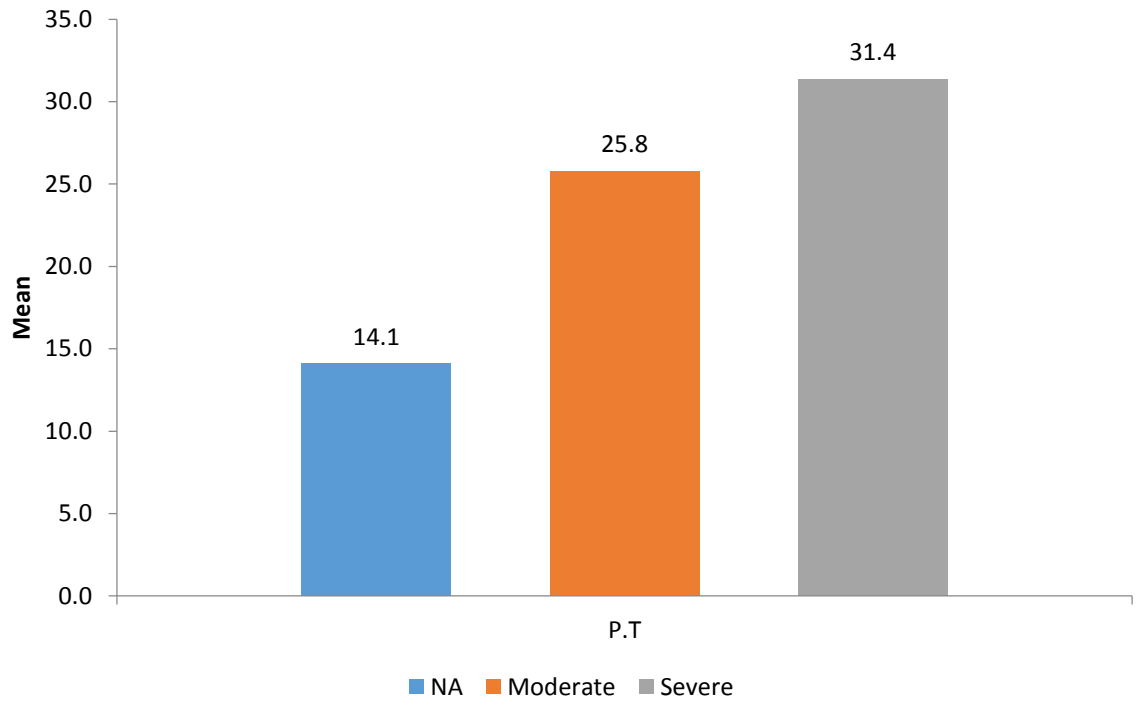


Figure: 44

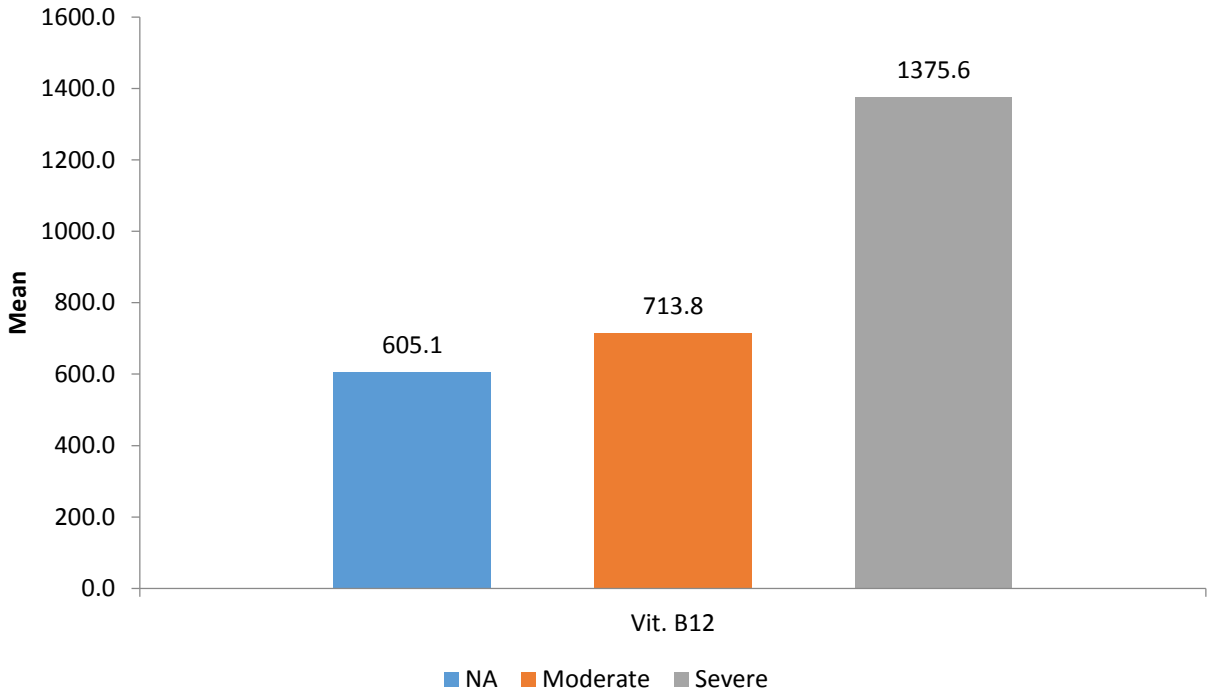
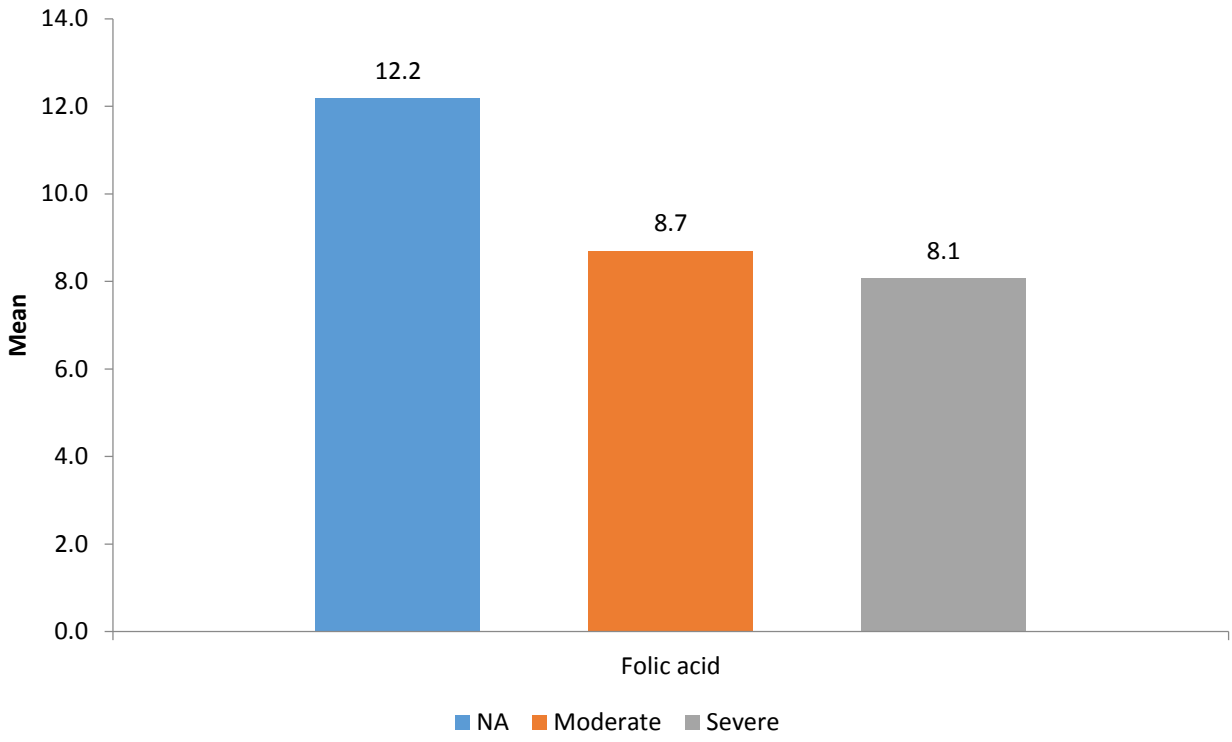


Figure: 45



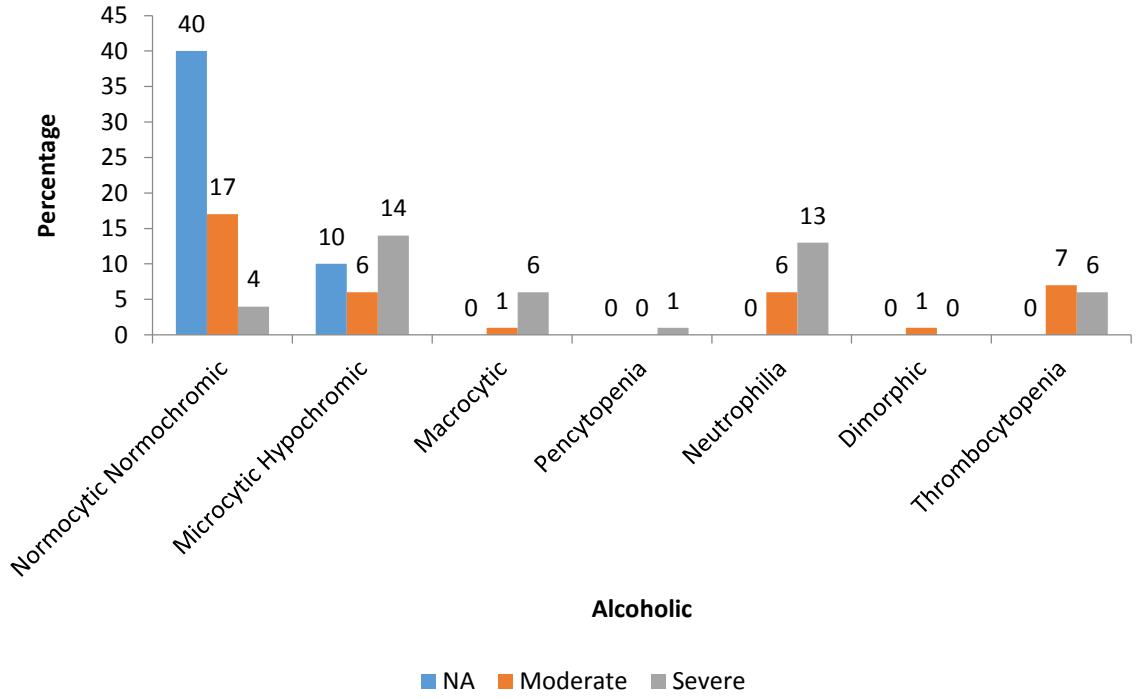
Inference:

In our study, mean P.T is 25.76 ±6.41 in moderate alcoholics and 31.35 ± 5.39 in severe alcoholics. Mean Folate level is 8.71 ± 1.00 in moderate drinkers and 8.08 ±1.01 in severe drinkers. Mean serum vitamin B12 is 713.80 ± 374.14, in moderate drinkers and 1375.60 ± 510.03 in severe drinkers. (p-value is <0.001) which is significant.

Table: 21

		Alcoholic			Total	Chi square	p
		NA	Moderate	Severe			
Normocytic Normochromic	No	10	8	21	39	29.38	0.000
	Yes	40	17	4	61		
Microcytic Hypochromic	No	40	19	11	70	10.86	0.004
	Yes	10	6	14	30		
Macrocytic	No	50	24	19	93	15.21	0.000
	Yes		1	6	7		
Pencytopenia	No	50	25	24	99	3.03	0.220
	Yes			1	1		
Neutrophilia	No	50	19	12	81	29.82	0.000
	Yes		6	13	19		
Dimorphic	No	50	24	25	99	3.03	0.220
	Yes		1		1		
Thrombocytopenia	No	50	18	19	87	15.12	0.001
	Yes		7	6	13		
Total		50	25	25	100		

Figure: 46



Inference:

In our study, 40% of alcoholics are having microcytic, hypochromic blood picture, macrocytosis is seen in 14% of patients. Pancytopenia and Dimorphic blood picture is seen in only 2% of alcoholics. 38% of alcoholics are having Neutrophilia due to infection, thrombocytopenia is seen in 26% of alcoholics. Even in non-alcoholics 20% of them are having Microcytic hypochromic blood picture.

Dimorphic and pancytopenia blood picture in alcoholics on statistical analysis shows ‘p’ value of 0.220 and the association is insignificant.

Table: 22 Abnormal RBC Morphology

		Alcoholic			Total	'p' value
		Moderate	**Severe**	**NA**		
Acanthocyte	Yes	1	5	0	6	0.015
	No	24	20	50	94	
Target Cell	Yes	6	12	0	18	<0.001
	No	19	13	50	82	
Schistocyte	Yes	1	4	0	5	0.030
	No	24	21	50	95	
Elliptocyte	Yes	1	5	0	6	0.015
	No	24	20	50	94	
Ovolocyte	Yes	2	5	0	7	0.000
	No	23	20	50	93	
Stomatocyte	Yes	4	8	0	12	<0.001
	No	21	17	50	88	
Total		50	25	25	100	

Figure: 47

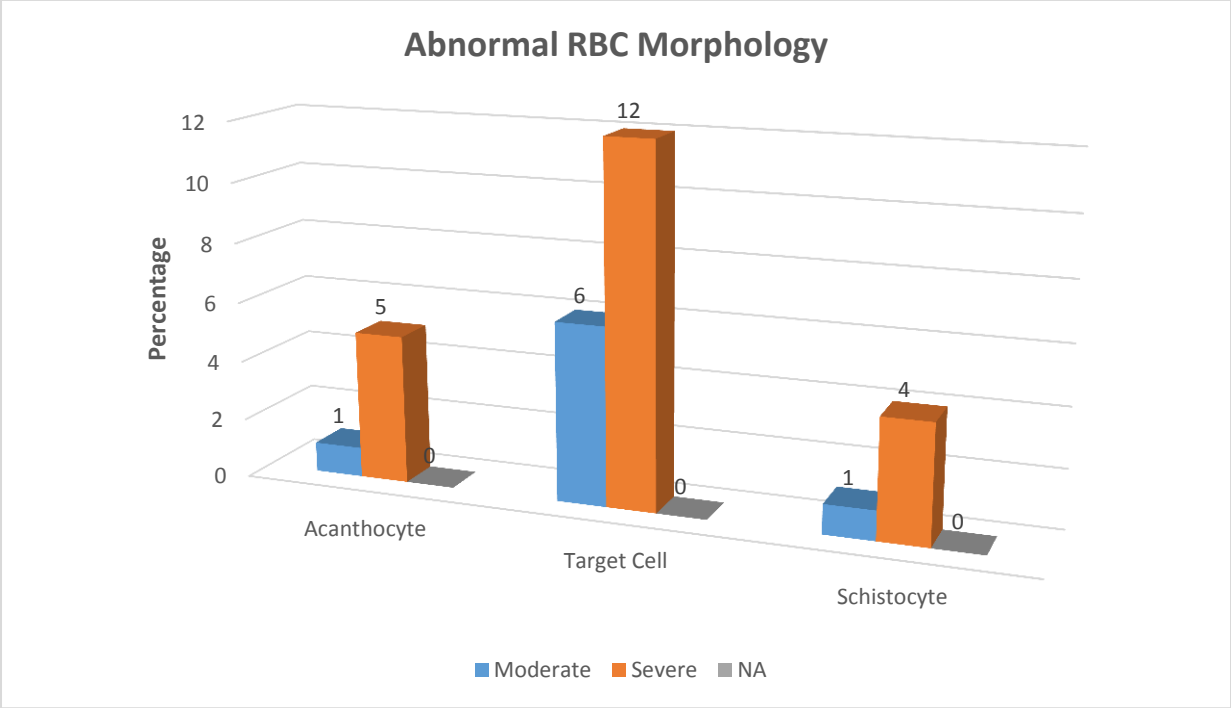
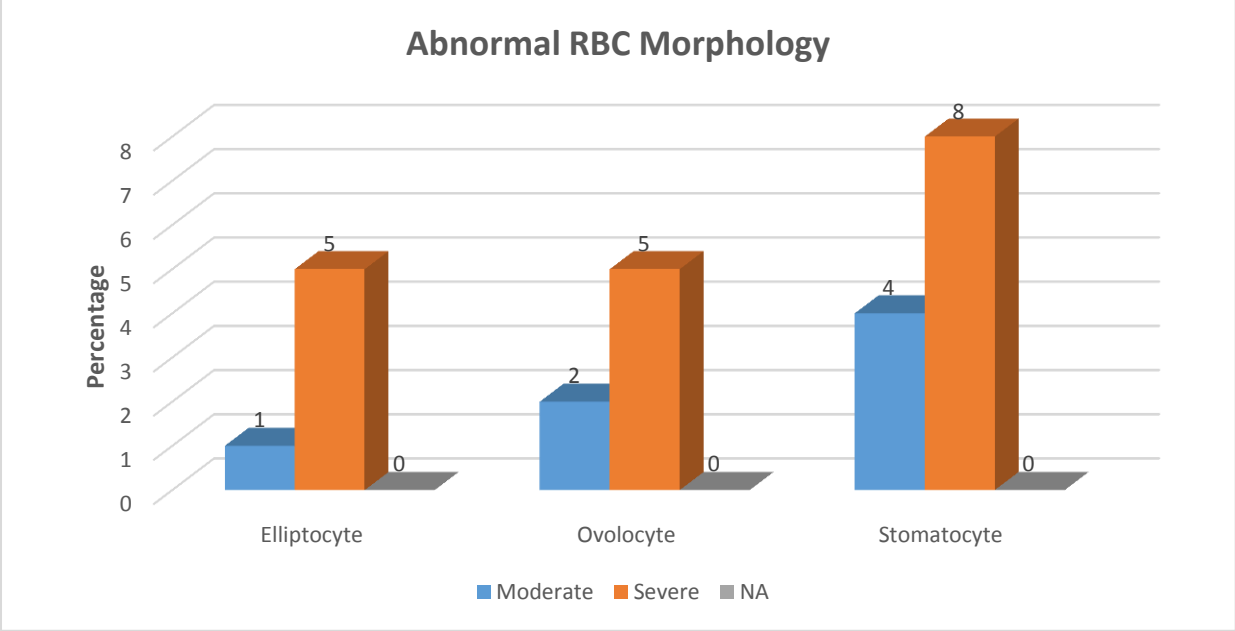


Figure: 48



Inference:

Target cells are seen in 36% of alcoholics in our study. Stomatocytes is 24% of alcoholics with p-value of <0.001.

Discussion

DISCUSSION

In our study the Hematological profile of moderate alcoholics, severe alcoholics and non-alcoholics are compared.

In our study the lowest age in the alcoholic group is 26 and highest age is 71.

Table: 23

	T.Oduola et al Study	**Our Study**	**D.Chalmers et al Study**
Mean Age in years	36.04 ± 11.28	35.68 ± 10.30	59.9

The maximum number of alcoholics are in the age group of 31 to 50 years and alcohol abuse less common below 25 years and above 71 years. Among 50 alcoholics 90% are Male and only 10% are Female.

Table: 24

Sex	T.Oduola et al	Ray R et al	Hislop et al	Our Study	D. Chalmers et al
Male	100%	100%	74.35%	90%	66.66%
Female	-	-	25.64%	10%	33.33%

This shows that alcoholism is more common in males than Females. But there is changing trend in the Females for alcohol consumption.

Among 50 alcoholics 78% of patients are in low socio economic status and 22% of them are in middle socio-economic status. This shows that poor people are severe alcoholic and they are consuming less quality drinks. They are suffering from more hematological manifestations.

In our study, 42% of alcoholics came with the complaints of pedal oedema and Ascites. Melena is present in 40% of patients Jaundice in 30%, Hematemesis in 20% of patients. 4% of alcoholics are presented with peripheral Neuropathy and 2% with cerebellar ataxia.

Duration of alcohol intake in our study is 42% of patients are consuming for 11-20 years, 36% of them are consuming for more than 20 years and 22% of patients consuming for less than 10 years.

Table: 25

Duration	**D.Chalmers et al**	**T.Oduola et al**	**Our Study**
1-10 years	-	50%	22%
11-20 years	58.71%	50%	42%
> 20 years	-	-	36%

Table: 26

	T.Oduola et al		Our Study		
	Moderate Alcoholics	**Severe Alcoholic**	**Moderate Alcoholics**	**Severe Alcoholic**	**Non Alcoholic**
Hb (gms%)	14.5 ± 1.20	14.8 ± 1.20	9.33 ± 1.20	9.37 ± 2.30	11.12 ± 2.24
WBC (Cells / mm^3)	4516.7 ± 2825.6	4733.3 ± 1400.6	7872 ± 3537.72	11072 ± 4286.17	8296 ± 1762.30
RBC	4.8999 ± 0.49	4.997 ± 0.503	3.26 ± 0.74	3.13 ±0.68	3.90 ± 0.58
PCV	44.2 ± 3.7	45.3 ± 3.8	33.55 ± 4.26	28.30 ± 6.14	33.55 ± 4.26
Platelet count	2.117 ±0.499	2.179 ± 0.417	1.63 ± 0.66	1.47 ± 0.67	2.50 ± 0.37
MCV (fl)	84.9 ± 9.1	89.7 ± 9.7	85.60 ± 8.35	93.44 ± 12.21	89.56 ± 3.84
MCH	28.4 ± 4.1	28.9 ± 4.3	25.48 ± 1.62	28.91 ± 3.18	30.54 ± 2.27
MCHC	32.3 ± 1.8	32.4 ± 1.8	32.57 ± 1.94	33.24 ± 2.00	36.18 ± 1.92
P.T	13.7 ± 1.3	13.2 ± 1.3	25.76 ± 6.41	31.35 ± 5.39	14.08 ± 0.83
T. Protein	7.5 ± 0.41	7.6 ± 0.37	6.68 ± 0.39	6.92 ± 0.37	7.44 ± 0.46
Albumin	4.5 ± 0.23	4.6 ±0.21	3.55 ± 0.31	3.40 ± 0.28	4.44 ± 0.51
SGOT	7.9 ± 0.43	12 ± 0.9	67.40 ± 27.45	94.44 ± 50.51	20.16 ±4.11
SGPT	7.3 ± 0.42	10.7 ± 0.81	48.84 ± 15.33	57.44± 21.27	27.40 ± 5.08
Alk. Phosphatase	95 ± 4.10	124 ± 3.69	48.16 ± 8.28	54.52 ± 10.89	51.92 ± 10.47

Table: 27

	DAS SK, Mukherjee S, Vasudevan DM, Balakrishnan V Study	**Our Study**		
		Moderate Alcoholics	**Severe Alcoholic**	**Non Alcoholic**
Hb (gms%)	13.6 ± 3.19	9.33 ± 1.20	9.37 ± 2.30	11.12 ± 2.24
WBC (Cells / mm^3)	7800 ± 4000	7872 ± 3537.72	11072 ± 4286.17	8296 ± 1762.30
RBC	4.6 ± 0.99	3.26 ± 0.74	3.13 ± 0.68	3.90 ± 0.58
PCV	37.5 ± 8.33	33.55 ± 4.26	28.30 ± 6.14	33.55 ± 4.26
Platelet count	2.05 ± 0.66	1.63 ± 0.66	1.47 ± 0.67	2.50 ± 0.37
MCV (fl)	82.2 ± 5.30	85.60 ± 8.35	93.44 ± 12.21	89.56 ± 3.84
MCH	29.8 ± 2.69	25.48 ± 1.62	28.91 ± 3.18	30.54 ± 2.27
MCHC	36.3 ± 2.15	32.57 ± 1.94	33.24 ± 2.00	36.18 ± 1.92
P.T	1.2 ± 0.17	25.76 ± 6.41	31.35 ± 5.39	14.08 ± 0.83

In our study, Hb level is less than 10 in 72 % and 76% of moderate and severe alcoholic respectively. In non-alcoholics, only 24% of patient's Hb level is less than 10 gms. WBC count in moderate alcoholics is less when compared with control group. Severe alcoholics have increased WBC count than control, due to infections. This shows that alcoholics are more prone for infections. MCV in severe alcoholics is high (93.44 ± 12.21). so macrocytosis is common in alcoholics and it indicates severity of alcoholism.

Thrombocytopenia is present in 24% of severe alcoholics and 28% of moderate alcoholics. 74% of alcoholics are Anemic.

Our study showed predominantly microcytic hypochromic Anemia (56% in severe, 24% in moderate alcoholic). Macrocytosis is seen in 14% of alcoholics. Pancytopenia in 2% of alcoholics, Dimorphic blood picture in 2% of alcoholics. Thrombocytopenia is seen in 26% of alcoholics.

Abnormal RBC morphology like target cells, Stomatocyte, Acanthocyte are present in 36%, 24%, 12% of alcoholics.

Hypoalbuminemia is seen in alcoholics in our study. SGOT is more than SGPT in our study. Alkaline phosphatase has no significance (p-value is 0.085). Blood urea, Serum Creatinine and P.T. is elevated. Serum

Folic acid is low when compared with controls. Vitamin B_{12} is high in alcoholics especially in severe alcoholics.

Conclusion

CONCLUSION

- ✓ Alcoholism is present in both males and females and predominantly seen in males.
- ✓ Alcoholism is common in lower socio-economic status people, especially in middle aged people 3^{rd} to 5^{th} decade.
- ✓ Anemia is most common among chronic drinkers. Anemia severity is related to duration and amount of alcohol consumption.
- ✓ Microcytic hypochromic blood picture is most common followed by macrosytosis.
- ✓ Severe alcoholics are more prone for infections.
- ✓ Thrombocytopenia is also a feature of alcoholics and is more common in severe drinkers.
- ✓ Macrocytosis in alcoholics is due to Folic acid deficiency not due to Cobalamin deficiency.
- ✓ Vitamin B_{12} level and prothrombin time is elevated in chronic alcoholics, especially in severe drinkers.
- ✓ Target cells stomatocytes, acanthocytes seen in peripheral smear suggest that hemolysis is also a cause of anemia in alcoholics.
- ✓ Macrocytosis is due to effect of alcohol on Bone marrow and Folate deficiency.

- ✓ Chronic Gastro intestinal bleeding in alcoholics cause Iron deficiency and leads to microcytic hypochromic anemia.
- ✓ Detection of hematological changes in alcohol abuse early, and treating them will prevent alcohol related complications and decrease the morbidity and mortality in alcoholics.
- ✓ Detection of hematological changes in chronic alcoholics and giving psychiatric counselling and treatment for alcohol dependence will decrease the future complications like Cirrhosis Liver, Cardiac and Renal Disease, Cerebellar degeneration, Neuropathy, Pancreatitis, etc. and reduce the morbidity and mortality in alcoholics.

Summary

SUMMARY

Alcoholism is strongly associated with anemia. The present study was done to compare the Haematological changes in moderate and severe drinkers and with non-alcoholics. With inclusion and exclusion criteria, patients were carefully selected and demographic data, history, clinical and laboratory data were evaluated after ethical committee clearance and consent from the patient.

In study group, 74% of alcoholics are Anemic which is higher significantly than the control group, in which 24% of them are Anemic. Macrocytosis is seen in 18% of alcoholics and it is statistically significant when compared with control groups in which no one has macrocytosis.

Total count in severe alcoholics is significantly higher than control groups. This shows that alcoholics are more prone for infections, especially severe drinkers. RBC count also significantly lower in alcoholics than controls. Thrombocytopenia in alcoholics is highly significant in our study.

Low Folic acid in alcoholics also contributes to the etiology of macrocytic blood picture. Structurally abnormal RBC's in peripheral smear shows that alcohol directly affect the erythropoiesis. Serum vitamin B_{12} is high in alcoholics significantly when compared with controls. This shows that macrocytic Anemia in alcoholics is always due to folate deficiency and not due to Cobalamin deficiency.

In the present study 42% of alcoholics has microcytic, hypochromic blood picture in the peripheral smear and it is highly significant than controls, in which only 24% shows the same blood picture. Pancytopenia and dimorphic picture is present in only 2% of alcoholics and it is insignificant.

Since microcytic hypochromic picture is common, the most common cause of anemia in alcoholics in our study is iron deficiency due to either nutritional (or) chronic gastro intestinal blood loss. Next common cause is macrocytic anemia due to Folate deficiency. Neutrophilia is seen in 38% of alcoholics due to infections.

These hematological changes in alcoholics are seen before the development of liver cell injury. So diagnosis at correct time and treating them will helps to prevent future complications like cardiac failure, Cirrhosis liver, and Neurological problems like cerebellar degeneration and peripheral Neuropathy, psychiatric counselling and treatment for alcohol dependence in these patients will help to decrease the morbidity and mortality related to alcohol induced organ damages.

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Proforma

Annexure

PROFORMA

HAEMATOLOGICAL MANIFESTATIONS OF ALCOHOLIC IN

COMPARISION WITH NON ALCOHOLICS

Name :

Age/Sex : IP No.:

Address: Occupation: DOA:

Social Economic Status:

PRESENTING COMPLIANTS:

Jaundice: Duration

Pain Abdomen Duration Site Aggravated by

Nature Radiation Relieved by

Distention of Abdomen

Vomiting Dyspepsia

Haemetemesis

Malena

Sensorium: Normal Altered

Fever: Duration

Nature: Continuous Remittent Intermittent

Degree: Mild Moderate Severe

Chills

Drug Intake

High Risk Behavior

Other Features

Alcoholic History:

A. Duration of Alcohol Consumption :

B. Quantity of Alcohol Consumption :

Past History:

Jaundice:

Transfusions: Vaccinations Injection

Drug Intake:

Family History:

Personal History:

Appetite	Normal	Decreased	
Diet	Vegetarian	Mixed	
Sleep	Normal	Decreased	
Bladder	Urine	Colour	Amount
Bowel	Colour	Frequency	
Habits Smoking	Tabacco	Chewing	
Sexual Dysfunction			
Menstrual & Obstetric History			

General Physical Examination:

Appearance	Healthy	Ill
Built	Well	Moderate
Poor		
Nourishment	Well	Moderate
Poor		
Weight (kgs)		
Height (cm)		
Pallor	Cyanosis	Lymphadenopathy
Icterus	Pedal Edema	Clubbing

Signs of Hepato Cellular Failure

Gynaecomastia

Parotid swelling Spider naevei Palmar erythema

Breast Atrophy

Testicular atrophy Dupuytrens contracture

Loss of axillary hair/pubic hair Asterixis

Jugular Venous Pressure

Vitals Temperature Pulse Blood Pressure

Respiration

Systemic Examination:

Per Abdomen Findings

Inspection

Shape Scaphoid Distended Visible Veins

Direction of flow

Umbilicus Peristalsis

Palpation Soft Tenderness Guarding Rigidity

Hepatomegaly Size

Surface

Margin

Consistency

Tenderness

Spleenomagaly Size

Surface

Margin

Consistency

Tenderness

Fluid Thrill

Abdominal girth

Percussion Shifting Dullness

Liver Dullness

Auscultation Bowel Sounds

Central Nervous System:

Sensorium Alert Stuporous Coma

Delirious

Cranial Nerves

Motor System

Sensory System

Cerebellar System
Other Findings

Respiratory System:
Trachea

Bilateral Air Entry

Character of breath sounds

Added sounds

Other Findings

Cardiovascular System:

Jugular Venous Pressure

Apical Impulse

Heart Sounds Normal Murmurs

Other Findings

Investigations:

Complete Blood Count:

Hemoglobin

MCV MCH MCHC PCV

Total count

Differential count P…….% L…….% M…….% E…….%

B……..%

Platelets

Peripheral Blood Smear

Random Blood Sugar:

L iver Function Test:

Total Bilirubin Direct

Total Proteins Alubumin

Aspartate amino transferase

Alanine amino transferase

Alkaline Phosphatase

Coagulation Profile:

Renal Function Test:

Ultrasound Abdomen:

Upper Gastro Intestinal Endoscopy:

Others:

Final Diagnosis:

Treatment:

Follow up:

Master Chart

S.No.	Name	Age	Sex	I.P. No.	S.E. Status	Moderate / Severe Alcoholic	Duration	Jaundice	Pedal Oedema	Haematemesis	Malena	Abdominal Distension	Altered Sensorium	Pallor	Icterus	Ascites	PE	Other Signs of Liver Cell Failure	Hepatomegaly	Splenomegaly	HSP	Hepatomegaly + Ascites
1	KANNAN	50	M	57370	L	S	>20 Yrs	N	Y	N	Y	Y	N	Y	N	Y	Y	Y	N	Y	N	N
2	KANDHASAMY	45	M	40930	L	S	>20 Yrs	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	N	N
3	SENGODAN	53	M	51308	L	S	>20 Yrs	Y	Y	N	Y	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y
4	GOVINDHAMMAL	45	F	63644	L	S	15 Yrs	Y	Y	N	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	Y
5	MADHESH	42	M	10250	M	S	15 Yrs	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	N	N
6	SREENIVASAN	49	M	59576	L	S	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
7	SELVAM	55	M	53712	L	S	>20 Yrs	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	Y	N	Y	N	N
8	ANNAMALAI	46	M	30180	M	S	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
9	USSEN	47	M	13149	L	S	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
10	SENDHAPPAN	51	M	51922	L	S	>20 Yrs	Y	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	N	Y	N	N
11	SUBRAMANIAM	49	M	43376	L	S	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
12	SHANMUGAM	47	M	2514	L	S	15 Yrs	Y	Y	N	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	Y
13	VENKATAPPA	48	M	41550	L	S	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
14	VEDIGOUNDER	50	M	53420	L	S	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
15	SEKAR	45	M	35786	L	S	>20 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
16	SURESH KUMAR	54	M	24968	L	S	<10 Yrs	N	Y	N	Y	Y	Y	Y	N	Y	Y	Y	N	Y	N	N
17	MOHAN	52	M	58806	L	S	<10 Yrs	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
18	MOHAMMED SATIFF	56	M	32764	M	M	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
19	MADHAIYAN	62	M	58700	L	S	>20 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
20	THALAI MALAI	57	M	59058	M	M	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
21	BALA KRISHNAN	55	M	3488	M	M	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
22	PALANISAMY	42	M	60932	L	S	>20 Yrs	N	Y	N	Y	Y	Y	N	N	Y	Y	N	N	Y	N	N
23	SIVAKUMAR	38	M	51882	M	M	<10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
24	RAJA	37	M	35696	M	M	<10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

S.No.	Splenomegaly + Ascites	Hbgm%	RBC Million/cmm	PCV %	MCV fl	MCH pg	MCHC %	T.C cells / cmm	Platelet Lakhs / cmm	T. Bilirubin mg/dl	D. Bilirubin mg /dl	Indirect Bilirubin mg /dl	T. Protein gm / dl	Albumin gm/dl	SGOT IU/L	SGPT IU / L	Alk. Phosphatas IU/L	RBS mg/dl	Blood Urea mg/dl	Serum Creatinine mg/dl	P.T sec.	Vit. B12 pg/ml	Folic acid ng/mm	P/s Study						
																								Normocytic Normochromic	Microcytic Hypochromic	Macrocytic	Pencytopenia	Neutrophilia	Dimorphic	Thrombocytopenia
1	Y	7.8	2.79	23.6	85	28.0	33.1	5000	1.3	2.4	0.8	1.6	7.0	3.4	54	38	51	260	96	1.8	26.0	>2000	6.25	N	Y	N	N	N	N	Y
2	Y	8.4	3.20	24.7	84	28.2	33.6	16800	1.0	7.6	2.5	5.1	6.8	3.2	96	54	42	82	92	1.6	36.0	1496	7.00	N	Y	N	N	Y	N	N
3	Y	15.6	4.86	43.0	89	32.1	36.3	7000	1.8	8.2	2.7	5.5	7.2	3.0	160	88	52	110	120	2.4	38.0	1128	8.10	Y	N	N	N	N	N	N
4	Y	7.0	2.70	23.0	76	24.6	29.2	12500	1.2	8.3	2.8	5.5	6.3	3.1	186	90	48	320	76	1.8	26.0	1863	6.90	N	Y	N	N	Y	N	N
5	Y	8.2	3.10	24.6	82	26.5	33.2	11800	1.4	6.4	2.2	4.2	6.6	3.3	170	92	56	96	72	1.6	24.0	1923	7.50	N	Y	N	N	Y	N	N
6	N	8.0	2.90	24.7	84	27.2	30.4	14100	1.0	1.8	0.6	1.2	7.0	3.5	65	42	50	110	76	1.4	22.0	1264	8.20	N	Y	N	N	Y	N	N
7	Y	10.6	3.19	31.0	97	33.2	34.2	9300	1.7	5.6	1.2	4.4	7.1	3.7	120	78	46	126	146	5.8	30.0	>2000	7.90	N	Y	N	N	N	N	N
8	N	6.9	2.39	23.4	78	24.6	33.0	12800	1.2	1.8	0.6	1.2	7.2	3.8	40	41	52	122	90	1.7	35.0	1227	8.40	N	Y	N	N	Y	N	Y
9	N	9.2	3.20	26.2	86	27.2	32.6	11600	1.0	1.2	0.4	0.8	7.3	3.9	48	50	56	117	82	1.6	36.0	1321	9.00	N	Y	N	N	N	N	Y
10	Y	8.4	2.75	25.2	92	30.5	33.3	2900	0.4	8.4	2.8	5.6	6.5	3.2	150	80	76	90	160	6.2	65.5	1392	8.30	N	N	N	Y	N	N	N
11	N	9.1	3.00	25.0	110	30.2	34.2	10800	0.9	1.8	0.6	1.2	7.2	3.7	80	46	50	78	93	1.7	32.0	1192	10.60	N	N	Y	N	N	N	Y
12	Y	8.9	2.85	26.0	112	27.4	33.2	9600	1.6	5.2	1.7	3.5	7.0	3.5	96	48	52	85	96	1.6	30.0	1921	9.70	N	N	Y	N	N	N	N
13	N	8.0	2.45	25.6	108	27.3	31.6	11700	1.4	1.2	0.4	0.8	6.8	3.7	56	28	42	90	42	1.2	28.0	1226	8.30	N	N	Y	N	Y	N	N
14	N	7.9	2.50	23.6	116	24.8	28.0	12600	1.8	1.6	0.7	0.9	7.2	3.5	46	20	72	120	50	1.0	34.0	1193	9.20	N	N	Y	N	Y	N	N
15	N	13.0	4.35	36.0	83	29.9	36.1	11800	4.0	1.8	0.6	1.2	7.2	3.7	42	36	47	72	120	4.2	36.0	529	7.20	Y	N	N	N	Y	N	N
16	Y	9.0	3.25	35.0	110	26.0	31.9	9800	1.8	2.6	0.9	1.7	6.8	3.5	72	58	47	110	92	2.0	38.0	1462	6.25	N	N	Y	N	N	N	N
17	N	8.2	3.30	24.7	75	24.8	33.2	10000	1.5	1.2	0.4	0.8	6.9	3.2	50	47	68	140	50	1.6	28.0	478	6.90	N	Y	N	N	Y	N	N
18	N	11.0	4.00	35.0	84	27.2	35.8	8400	1.8	1.6	0.5	1.1	7.0	3.6	56	56	42	130	58	1.4	30.0	468	8.10	Y	N	N	N	N	N	N
19	N	8.6	3.15	34.2	112	25.0	33.4	9200	1.5	1.2	0.4	0.8	8.1	3.8	58	58	40	126	70	1.5	32.0	481	8.30	N	N	Y	N	N	N	N
20	N	10.8	3.85	34.6	85	25.2	33.1	12600	1.7	1.1	0.4	0.7	7.2	3.7	60	60	36	128	96	4.6	34.0	620	7.90	Y	N	N	N	Y	N	N
21	N	10.2	3.95	34.4	84	25.7	34.2	8600	1.2	1.6	0.6	1.0	6.8	3.9	62	70	38	78	110	3.8	36.0	593	8.40	Y	N	N	N	N	N	N
22	Y	13.6	4.30	41.7	97	31.6	32.6	9100	1.2	2.6	0.8	1.8	6.6	3.2	82	58	72	83	96	5.8	25.4	464	9.00	Y	N	N	N	Y	N	N
23	N	11.2	4.20	38.0	83	25.8	35.6	9200	1.4	1.8	0.6	1.2	6.7	3.8	42	66	35	92	96	2.0	26.0	523	8.30	Y	N	N	N	N	N	N
24	N	7.6	2.00	34.0	75	24.3	30.8	5600	0.5	1.2	0.4	0.8	6.9	4.2	47	72	41	102	50	1.3	28.0	493	10.60	N	N	N	N	N	Y	Y

S.No.	Name	Age	Sex	I.P. No.	S.E. Status	Moderate / Severe Alcoholic	Duration	Jaundice	Pedal Oedema	Haematemesis	Malena	Abdominal Distension	Altered Sensorium	Pallor	Icterus	Ascites	PE	Other Signs of Liver Cell Failure	Hepatomegaly	Splenomegaly	HSP	Hepatomegaly + Ascites
25	RUCKMANI	62	F	4141	L	M	>20 Yrs	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	N	N
26	RAJENDRAN	65	M	22868	L	M	>20 Yrs	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	Y	N	Y	N	N
27	MADESHWARAN	58	M	6515	L	M	>20 Yrs	Y	Y	N	Y	Y	N	Y	Y	Y	Y	Y	N	Y	N	N
28	DHANABAGYAM	62	F	9521	L	M	>20 Yrs	Y	Y	N	Y	Y	N	Y	Y	Y	Y	Y	N	Y	N	N
29	SANKAR	48	M	20805	L	S	>20 Yrs	N	Y	N	N	Y	N	N	N	Y	Y	Y	Y	N	N	Y
30	YOGARAJAN	35	M	71230	L	M	<10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
31	MUTHU	71	M	21001	L	S	>20 Yrs	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	Y	N	N	N	N
32	SUBRAMANI	38	m	20995	l	m	<10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
33	CHANDRASEKAR	39	M	67908	L	M	<10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
34	VIJAY BABU	40	M	39054	M	M	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
35	SURESH KUMAR	43	M	30678	L	S	>20 Yrs	Y	Y	N	Y	Y	N	Y	Y	Y	Y	Y	N	Y	N	N
36	SUMATHI	37	F	67966	M	M	<10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
37	VIJAYA KUMAR	36	M	18325	L	M	<10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
38	SAKTHIVEL	38	M	54308	L	M	<10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
39	SURYA	35	M	4989	L	M	<10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
40	HARIDAS	36	M	51922	L	M	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
41	PHILIP	37	M	53420	L	M	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
42	KUMARESAN	38	M	50814	L	M	15 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
43	KULANTHAIAMMAL	50	F	40069	L	S	>20 Yrs	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	N	N
44	KRISHNAN	28	M	40350	M	M	10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
45	PERUMAL	55	M	40710	L	S	>20 Yrs	N	Y	Y	Y	Y	N	Y	N	Y	Y	Y	N	Y	N	N
46	KAMATCHI	27	M	30200	M	M	10 Yrs	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
47	MAHALI	36	M	37250	L	M	15 Yrs	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	Y	N	N	Y	N
48	RAJA	29	M	35786	L	M	10 Yrs	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	N

S.No.	Splenomegaly + Ascites	Hbgm%	RBC Million/cmm	PCV %	MCV fl	MCH pg	MCHC %	T.C cells / cmm	Platelet Lakhs / cmm	T. Bilirubin mg/dl	D. Bilirubin mg /dl	Indirect Bilirubin mg /dl	T. Protein gm / dl	Albumin gm/dl	SGOT IU/L	SGPT IU / L	Alk. Phosphatas IU/L	RBS mg/dl	Blood Urea mg/dl	Serum Creatinine mg/dl	P.T sec.	Vit. B12 pg/ml	Folic acid ng/mm	P/s Study						
																								Normocytic Normochromic	Microcytic Hypochromic	Macrocytic	Pencytopenia	Neutrophilia	Dimorphic	Thrombocytopenia
25	Y	6.8	2.15	33.0	78	24.2	28.6	6200	0.8	7.6	2.5	5.1	7.0	3.2	149	78	56	103	60	1.4	30.0	1423	9.70	N	Y	N	N	N	N	Y
26	Y	8.2	2.75	36.0	76	23.8	32.4	4800	1.2	8.2	2.8	5.4	6.2	3.4	143	76	58	90	72	1.6	32.0	1236	9.20	N	Y	N	N	N	N	N
27	Y	11.2	3.80	38.0	83	26.8	35.2	5200	1.6	6.8	2.3	4.5	6.5	3.0	90	52	64	78	50	1.0	28.0	1391	8.30	Y	N	N	N	N	N	N
28	Y	7.0	2.25	34.0	80	24.7	29.6	6000	0.8	5.9	1.9	4.0	6.3	3.5	110	72	66	68	48	1.2	38.0	1263	6.20	N	Y	N	N	N	N	Y
29	N	14.3	4.36	38.6	89	32.8	37.0	26200	1.5	2.6	0.9	1.7	6.8	3.6	96	52	61	102	89	4.9	42.0	1272	8.30	Y	N	N	N	Y	N	N
30	N	10.2	3.85	36.0	86	25.2	32.3	11800	1.3	1.8	0.6	1.2	7.0	3.0	56	29	38	92	62	1.3	16.0	523	7.20	N	Y	N	N	Y	N	N
31	Y	10.1	3.49	29.9	86	28.9	33.8	7600	2.3	7.2	2.2	5.0	6.6	3.2	170	86	70	90	102	3.6	15.6	>2000	7.60	N	Y	N	N	N	N	N
32	N	9.2	3.65	36.2	82	25.4	30.6	4800	1.4	1.2	0.4	0.8	7.2	3.2	52	37	42	230	110	3.2	30.0	526	9.30	Y	N	N	N	N	N	N
33	N	9.8	3.75	36.4	78	24.7	30.8	5600	2.3	1.1	0.4	0.7	7.1	3.3	48	38	42	240	90	2.8	18.0	492	9.10	N	Y	N	N	N	N	Y
34	N	9.6	3.70	36.2	78	24.8	32.6	7800	2.8	1.6	0.7	0.9	6.8	3.4	52	40	40	110	86	2.2	20.0	593	8.80	Y	N	N	N	N	N	N
35	Y	8.2	2.47	23.7	96	33.2	34.6	9500	1.5	6.8	2.2	4.6	6.5	3.0	210	96	72	92	110	4.8	31.7	1098	8.60	N	Y	N	N	N	N	Y
36	N	10.0	3.85	29.6	86	25.4	33.4	5200	1.8	1.8	0.6	1.2	6.5	3.5	56	28	47	112	88	2.0	20.0	478	8.75	Y	N	N	N	N	N	N
37	N	9.6	3.78	36.0	88	26.2	32.6	4100	2.6	1.2	0.4	0.8	6.2	3.7	62	34	46	116	79	1.6	22.0	482	9.25	Y	N	N	N	N	N	N
38	N	9.8	3.80	36.4	90	25.8	32.8	4000	2.8	1.1	0.4	0.7	6.4	3.8	49	38	50	86	82	1.8	16.0	592	10.65	Y	N	N	N	N	N	Y
39	N	10.4	3.95	36.6	86	26.4	33.4	4600	3.0	1.3	0.5	0.8	6.6	3.9	57	42	52	92	68	1.3	18.0	612	8.20	Y	N	N	N	N	N	N
40	N	9.2	4.00	36.8	110	23.8	36.2	8600	1.0	1.1	0.4	0.7	6.0	4.0	58	38	50	79	70	1.1	20.0	623	7.90	Y	N	N	N	N	N	Y
41	N	8.8	3.00	36.0	78	24.7	33.8	9200	1.8	1.2	0.4	0.8	6.0	3.5	60	37	52	88	78	2.6	22.0	493	7.80	N	Y	N	N	N	N	N
42	N	8.4	2.85	36.2	108	24.2	30.2	4100	1.5	1.6	0.7	0.9	6.1	3.7	62	39	50	85	79	2.2	18.0	486	7.90	N	N	Y	N	N	N	N
43	Y	9.0	2.90	26.7	92	31.0	33.7	12700	1.5	7.2	2.2	5.0	7.0	3.2	78	62	45	76	112	5.2	28.0	>2000	8.35	N	Y	N	N	Y	N	N
44	N	8.2	2.10	26.0	86	26.2	30.8	11800	1.8	1.2	0.4	0.8	6.8	3.6	62	41	50	92	90	1.8	26.0	528	9.60	Y	N	N	N	Y	N	N
45	Y	8.5	2.58	24.8	96	32.9	34.3	9200	0.6	2.2	0.8	1.4	6.4	3.0	68	42	47	90	150	6.8	34.4	>2000	7.90	N	Y	N	N	N	N	Y
46	N	9.0	2.62	25.6	88	26.8	32.6	13800	1.9	1.1	0.4	0.7	6.9	3.8	56	44	52	96	86	1.6	32.0	474	9.80	Y	N	N	N	Y	N	N
47	N	9.3	2.63	26.0	90	31.6	32.8	14600	0.9	7.0	2.2	4.8	6..9	3.4	76	42	47	94	93	2.0	30.0	1782	9.20	Y	N	N	N	Y	N	Y
48	N	9.0	2.68	25.8	88	24.2	32.4	15000	1.2	1.6	0.7	0.9	7.0	3.5	58	45	56	110	99	3.0	26.0	528	9.00	Y	N	N	N	Y	N	N

S.No.	Name	Age	Sex	I.P. No.	S.E. Status	Moderate / Severe Alcoholic	Duration	Jaundice	Pedal Oedema	Haematemesis	Malena	Abdominal Distension	Altered Sensorium	Pallor	Icterus	Ascites	PE	Other Signs of Liver Cell Failure	Hepatomegaly	Splenomegaly	HSP	Hepatomegaly + Ascites
49	BASKAR	28	M	35787	L	M	10 Yrs	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	N
50	VELUSAMY	47	M	36997	L	S	>20 Yrs	N	Y	N	Y	Y	N	Y	N	Y	Y	Y	N	Y	N	N
51	AMMAKKA	60	F	110006	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
52	ARUMUGAN	61	M	109720	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
53	RAJA RATHINAM	64	M	169854	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
54	JAGAN	28	M	110336	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
55	SUNDARAM	29	M	110240	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
56	NALLAMMAL	62	F	297	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
57	PALANIAPPAN	52	M	616	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
58	MADHIALAGAN	29	M	106	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
59	RAJATHI	61	F	1300	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
60	PALANIVEL	54	M	1064	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
61	SADAIYAN	57	M	1216	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
62	SRIRANGAN	56	M	122132	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
63	SUNDARAMOORTHY	58	M	2374	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
64	IRUSAPPAN	53	M	2978	L	NA	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
65	MURUGAN	55	M	2968	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
66	PAPPATY	56	F	3094	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
67	AMMASI	58	M	4504	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
68	PERIYANNAN	57	M	4530	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
69	GOPAL	26	M	4302	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
70	VENKATESH	46	M	4816	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
71	RAJAMANIKAM	42	M	1202	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
72	ANSAR	44	M	10170	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

S.No.	Splenomegaly + Ascites	Hbgm%	RBC Million/cmm	PCV %	MCV fl	MCH pg	MCHC %	T.C cells / cmm	Platelet Lakhs / cmm	T. Bilirubin mg/dl	D. Bilirubin mg /dl	Indirect Bilirubin mg /dl	T. Protein gm / dl	Albumin gm/dl	SGOT IU/L	SGPT IU / L	Alk. Phosphatas IU/L	RBS mg/dl	Blood Urea mg/dl	Serum Creatinine mg/dl	P.T sec.	Vit. B12 pg/ml	Folic acid ng/mm	P/s Study						
																								Normocytic Normochromic	Microcytic Hypochromic	Macrocytic	Pencytopenia	Neutrophilia	Dimorphic	Thrombocytopenia
49	N	8.8	2.30	26.0	90	23.8	31.6	5200	1.6	1.2	0.4	0.8	7.1	3.2	62	47	54	130	92	2.2	28.0	623	8.50	Y	N	N	N	N	N	N
50	N	7.8	2.24	22.6	101	34.8	34.5	13200	1.7	1.9	0.7	1.2	6.8	3.2	68	46	49	88	110	4.8	26.0	1460	8.30	N	Y	N	N	Y	N	N
51	N	10.6	3.80	36.0	90	27.2	34.0	7200	2.2	0.8	0.3	0.5	7.0	3.8	15	20	50	106	19	0.8	13.0	481	11.20	N	Y	N	N	N	N	N
52	N	12.4	4.00	32.0	88	30.0	35.8	8400	2.0	1.2	0.3	0.9	6.8	4.0	18	25	48	110	20	1.0	15.2	486	9.60	Y	N	N	N	N	N	N
53	N	13.0	3.58	34.0	86	32.2	34.8	10600	2.4	0.6	0.2	0.4	7.2	4.5	20	30	60	120	23	1.1	14.6	464	10.12	Y	N	N	N	N	N	N
54	N	11.8	3.60	35.0	85	33.6	33.9	7800	2.2	1.1	0.4	0.7	7.5	3.6	16	22	65	160	16	1.2	12.2	620	9.60	Y	N	N	N	N	N	N
55	N	10.9	4.10	36.0	86	31.9	35.9	6900	2.3	1.2	0.5	0.7	7.6	4.3	22	26	72	180	18	6.0	12.4	640	8.90	Y	N	N	N	N	N	N
56	N	11.6	3.95	35.3	86	30.8	34.8	8200	2.3	1.0	0.3	0.7	8.0	4.2	18	37	76	202	24	0.8	13.0	670	12.60	Y	N	N	N	N	N	N
57	N	11.2	3.80	43.0	92	31.0	36.4	8400	2.4	0.8	0.3	0.5	7.8	4.2	15	20	46	92	19	1.2	13.6	492	12.00	Y	N	N	N	N	N	N
58	N	2.2	3.60	42.0	90	27.2	35.9	9000	3.1	1.2	0.3	0.9	8.2	3.8	17	23	48	110	30	1.1	14.2	620	13.20	Y	N	N	N	N	N	N
59	N	10.8	3.50	40.0	86	27.2	36.8	9200	2.6	0.6	0.2	0.4	8.5	3.9	18	22	49	130	32	0.6	15.2	860	9.30	Y	N	N	N	N	N	N
60	N	11.6	3.90	38.0	88	27.0	33.9	7800	2.3	1.1	0.4	0.7	6.9	3.8	20	23	50	150	20	0.8	13.0	483	12.30	Y	N	N	N	N	N	N
61	N	11.9	4.00	44.0	90	28.0	33.6	8600	2.1	1.2	0.5	0.7	7.2	4.6	22	24	52	138	26	1.3	14.8	620	12.60	Y	N	N	N	N	N	N
62	N	10.4	3.42	43.0	92	29.0	32.8	8800	2.1	1.0	0.3	0.7	7.4	5.0	16	26	40	146	24	0.8	12.6	626	13.40	N	Y	N	N	N	N	N
63	N	9.8	3.63	40.0	93	29.2	35.5	9400	2.5	1.1	0.4	0.7	7.6	5.2	17	28	42	192	28	0.7	13.0	530	16.20	N	Y	N	N	N	N	N
64	N	8.8	3.82	38.0	94	29.4	36.8	9300	2.9	0.9	0.3	0.6	7.8	5.3	18	30	44	106	20	0.6	13.2	535	17.10	N	Y	N	N	N	N	N
65	N	9.8	4.10	40.0	95	29.6	34.9	16000	2.8	0.8	0.4	0.4	8.0	5.4	20	22	46	110	29	0.5	14.0	670	10.30	Y	N	N	N	N	N	N
66	N	10.1	3.82	39.0	96	30.8	35.8	10600	2.8	1.2	0.3	0.9	7.2	5.5	22	18	48	111	32	0.9	15.0	726	11.30	Y	N	N	N	N	N	N
67	N	10.2	3.68	40.0	98	32.4	40.6	5800	2.9	0.7	0.2	0.5	7.3	5.0	24	20	50	113	36	0.8	15.2	543	10.80	Y	N	N	N	N	N	N
68	N	10.6	3.50	41.0	99	34.2	38.4	6900	1.9	1.1	0.4	0.7	7.4	5.2	20	24	52	105	34	0.4	14.6	492	12.30	Y	N	N	N	N	N	N
69	N	11.4	3.85	42.0	94	36.0	36.4	7000	1.9	0.8	0.3	0.5	7.8	4.8	17	26	70	104	39	0.6	14.8	463	9.80	Y	N	N	N	N	N	N
70	N	13.6	3.90	40.0	90	30.6	39.2	7200	1.5	1.2	0.3	0.9	7.6	4.7	18	28	78	106	38	0.7	14.7	723	9.90	Y	N	N	N	N	N	N
71	N	11.6	3.80	40.0	90	31.9	36.4	6800	2.5	0.8	0.3	0.5	7.6	4.2	17	20	40	90	19	0.8	15.0	464	11.30	Y	N	N	N	N	N	N
72	N	12.3	3.60	42.0	88	32.0	35.9	7200	2.6	1.2	0.3	0.9	7.5	4.4	18	24	46	106	30	0.6	15.2	530	15.60	Y	N	N	N	N	N	N

S.No.	Name	Age	Sex	I.P. No.	S.E. Status	Moderate / Severe Alcoholic	Duration	Jaundice	Pedal Oedema	Haematemesis	Malena	Abdominal Distension	Altered Sensorium	Pallor	Icterus	Ascites	PE	Other Signs of Liver Cell Failure	Hepatomegaly	Splenomegaly	HSP	Hepatomegaly + Ascites
73	MADESH	47	M	10250	L	NA	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
74	KARTHIKEYAN	48	M	10456	L	NA	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
75	ILAYAPERUMAL	44	M	7160	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
76	MUTHUSAMY	49	M	9264	L	NA	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
77	CHELLAMANI	42	M	2325	L	NA	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
78	RAJENDRAN	36	M	10810	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
79	ALLIMUTHU	32	M	9720	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
80	VARADHARAJ	37	M	3001	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
81	JEYARAMAN	38	M	12068	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
82	RAJU	39	M	3487	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
83	MARIAPPAN	36	M	14570	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
84	SHANMUGAM	37	M	3779	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
85	CHENNAIAPPAN	39	M	17376	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
86	THIRUMURUGAN	37	M	5009	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
87	SELVAM	38	M	21432	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
88	PERIYASAMY	32	M	20950	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
89	RAMAN	35	M	22456	L	NA	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
90	MANOHARAN	37	M	22238	L	NA	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
91	RAMASAMY	39	M	6749	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
92	NATARJAN	46	M	22840	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
93	PALANISAMY	43	M	6927	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
94	MURUGESAN	49	M	27580	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
95	KRISHNAN	46	M	26934	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
96	SHANMUGAM	49	M	9259	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

S.No.	Splenomegaly + Ascites	Hbgm%	RBC Million/cmm	PCV %	MCV fl	MCH pg	MCHC %	T.C cells / cmm	Platelet Lakhs / cmm	T. Bilirubin mg/dl	D. Bilirubin mg /dl	Indirect Bilirubin mg /dl	T. Protein gm / dl	Albumin gm/dl	SGOT IU/L	SGPT IU / L	Alk. Phosphatas IU/L	RBS mg/dl	Blood Urea mg/dl	Serum Creatinine mg/dl	P.T sec.	Vit. B12 pg/ml	Folic acid ng/mm	P/s Study						
																								Normocytic Normochromic	Microcytic Hypochromic	Macrocytic	Pencytopenia	Neutrophilia	Dimorphic	Thrombocytopenia
73	N	8.6	2.50	43.0	86	30.0	36.2	7600	3.2	0.6	0.2	0.4	7.8	4.6	19	26	48	180	26	0.4	13.6	623	14.30	Y	N	N	N	N	N	N
74	N	7.8	1.90	36.0	86	27.2	35.8	8200	2.8	1.1	0.4	0.7	7.2	4.8	20	28	50	202	25	1.1	13.8	720	13.80	Y	N	N	N	N	N	N
75	N	9.2	2.80	38.0	85	27.6	36..9	8400	2.2	1.2	0.5	0.7	7.4	5.0	22	30	56	210	20	1.2	13.9	900	12.90	N	Y	N	N	N	N	N
76	N	8.0	3.90	40.0	84	27.3	35.6	8600	2.8	1.0	0.3	0.7	7.6	3.8	20	32	52	212	26	0.9	13.0	430	11.60	N	Y	N	N	N	N	N
77	N	8.2	4.20	42.0	86	27.4	35.9	10000	2.8	1.1	0.4	0.7	7.5	3.9	17	30	54	160	24	0.8	14.0	486	10.80	Y	N	N	N	N	N	N
78	N	13.3	3.80	43.0	88	27.5	35.2	9200	2.6	0.9	0.3	0.6	7.8	3.7	18	28	56	175	30	0.7	14.2	660	11.40	Y	N	N	N	N	N	N
79	N	14.6	5.00	40.0	90	27.6	36.8	9600	2.7	0.8	0.3	0.5	6.9	3.8	20	30	58	178	32	0.8	14.4	663	12.00	Y	N	N	N	N	N	N
80	N	14.8	4.80	42.0	92	31.2	39.6	9800	2.9	1.2	0.3	0.9	7.0	4.0	24	32	62	179	36	0.9	14.6	662	13.00	Y	N	N	N	N	N	N
81	N	13.9	5.00	38.0	93	31.4	40.6	9400	2.3	0.7	0.2	0.5	7.2	4.2	26	36	64	60	34	0.4	14.8	592	13.10	Y	N	N	N	N	N	N
82	N	12.0	4.86	36.0	92	31.6	36.8	6800	1.9	1.1	0.4	0.7	7.4	4.4	28	28	68	80	38	0.8	15.0	468	12.20	Y	N	N	N	N	N	N
83	N	11.2	4.25	39.0	94	31.7	38.4	5200	2.1	0.8	0.3	0.5	7.9	4.6	30	30	62	82	20	0.7	13.8	760	14.30	Y	N	N	N	N	N	N
84	N	10.8	4.30	40.0	96	31.8	34.0	7600	2.2	1.2	0.3	0.9	7.2	4.7	32	30	66	86	21	0.9	13.9	823	10.90	Y	N	N	N	N	N	N
85	N	10.8	3.80	38.0	86	30.0	36.4	10600	2.4	0.8	0.3	0.5	6.8	4.6	15	20	28	110	19	0.6	13.6	466	11.30	Y	N	N	N	N	N	N
86	N	11.6	3.92	40.0	88	30.6	35.9	10800	3.1	1.2	0.3	0.9	7.0	4.2	17	26	30	116	26	0.7	14.0	430	12.60	Y	N	N	N	N	N	N
87	N	11.2	3.86	42.0	92	30.8	36.8	9200	3.2	0.6	0.2	0.4	7.2	3.9	19	21	40	111	30	0.8	14.2	560	13.20	N	Y	N	N	N	N	N
88	N	9.0	4.60	40.0	90	30.5	33.9	9600	2.6	1.1	0.4	0.7	8.0	3.8	20	30	42	112	32	1.1	12.9	624	12.90	N	Y	N	N	N	N	N
89	N	8.8	3.95	38.0	92	31.0	33.6	8200	2.2	1.2	0.5	0.7	8.2	4.0	22	32	46	98	34	1.2	12.8	735	11.30	Y	N	N	N	N	N	N
90	N	8.6	4.80	36.0	90	31.2	32.8	8600	2.5	1.0	0.3	0.7	8.3	4.2	24	34	48	106	36	0.6	14.2	846	12.60	N	Y	N	N	N	N	N
91	N	13.2	4.60	37.0	82	31.3	35.5	8000	2.6	1.1	0.4	0.7	8.4	4.4	26	26	50	109	38	0.5	14.2	935	13.20	N	Y	N	N	N	N	N
92	N	14.6	4.25	36.6	84	31.4	36.8	7600	2.7	0.9	0.3	0.6	7.6	4.6	17	28	52	108	30	0.4	14.4	824	12.90	Y	N	N	N	N	N	N
93	N	11.9	4.80	37.2	86	31.5	34.9	7800	2.7	0.8	0.3	0.5	6.9	4.8	15	30	46	180	28	0.6	14.6	520	11.80	Y	N	N	N	N	N	N
94	N	11.4	3.92	36.8	86	31.6	35.8	6200	2.7	1.2	0.3	0.9	7.0	4.9	19	32	44	200	29	0.8	14.7	464	10.60	Y	N	N	N	N	N	N
95	N	10.9	2.92	35.2	90	31.7	40.6	6400	2.7	0.7	0.2	0.5	7.2	5.0	20	34	48	160	30	1.0	14.8	532	11.90	Y	N	N	N	N	N	N
96	N	14.6	3.68	36.2	92	32.0	38.4	5900	2.9	1.1	0.4	0.7	7.3	5.2	26	36	50	192	19	1.1	14.9	492	12.80	Y	N	N	N	N	N	N

S.No.	Name	Age	Sex	I.P. No.	S.E. Status	Moderate / Severe Alcoholic	Duration	Jaundice	Pedal Oedema	Haematemesis	Malena	Abdominal Distension	Altered Sensorium	Pallor	Icterus	Ascites	PE	Other Signs of Liver Cell Failure	Hepatomegaly	Splenomegaly	HSP	Hepatomegaly + Ascites
97	GOPAL	44	M	2688	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
98	SIVALINGAM	45	M	27800	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
99	PANNEER	47	M	26996	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
100	MARIMUTHU	49	M	30344	L	NA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

S.No.	Splenomegaly + Ascites	Hbgm%	RBC Million/cmm	PCV %	MCV fl	MCH pg	MCHC %		Platelet Lakhs / cmm	T. Bilirubin mg/dl	D. Bilirubin mg /dl	Indirect Bilirubin mg /dl	T. Protein gm / dl	Albumin gm/dl	SGOT IU/L	SGPT IU / L	Alk. Phosphatas IU/L	RBS mg/dl	Blood Urea mg/dl	Serum Creatinine mg/dl	P.T sec.	Vit. B12 pg/ml	Folic acid ng/mm	P/s Study						
																								Normocytic Normochromic	Microcytic Hypochromic	Macrocytic	Pencytopenia	Neutrophilia	Dimorphic	Thrombocytopenia
97	N	13.8	4.20	36.4	90	29.0	36.4	5800	2.2	0.8	0.3	0.5	6.9	5.0	30	38	52	196	20	1.2	14.6	683	13.86	Y	N	N	N	N	N	N
98	N	14.0	3.86	35.2	86	29.6	39.2	6600	2.5	1.2	0.3	0.9	6.6	4.6	17	36	60	186	22	1.3	14.8	626	14.20	Y	N	N	N	N	N	N
99	N	11.6	3.80	38.0	86	35.6	36.4	7600	2.6	0.9	0.3	0.6	7.2	3.9	17	26	42	101	19	1.2	13.2	468	11.40	Y	N	N	N	N	N	N
100	N	11.0	3.90	36.0	90	35.8	35.9	8400	2.7	0.8	0.3	0.5	6.8	4.2	20	23	50	160	30	1.0	14.6	526	12.90	Y	N	N	N	N	N	N

Key to Master Chart

S.E. Stauts	-	Socio Economic Status
L	-	Low
M	-	Middle
Sex M	-	Male
F	-	Female
Alcoholic		
M	-	Moderate
S	-	Severe
PE	-	Pedal Oedema
HSP	-	Hepato Splenomegaly
RBS	-	Random Blood Sugar
P/S Study	-	Peripheral Smear Study
Y	-	Yes N - No
NA	-	Non-Alcoholic